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INTESTINAL TUBERCULOSIS

ITS IMPORTANCE, DIAGNOSIS AND TREATMENT

A STUDY OF THE SECONDARY ULCERATIVE TYPE

BY

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FOREWORD.

The present volume, the first one of a small series, represents the fruition of one of the ideals of Dr. E. L. Trudeau.

When he founded the Adirondack Cottage Sanatorium, forty years ago, while his major purpose was to cure cases of beginning tuberculosis, another and almost equally compelling and dominant impulse was to enquire into the nature of this mysterious disease with the aim that dominates all medical science, the aim to put the control of the disease into men's hands, and to enable them to protect themselves from it.

From the first, Dr. Trudeau published to the world the results of his observations and these were always of value. In the beginning his experiments were carried out in his own home and with crude apparatus, much of it manufactured in Saranac Lake by local artisans under his personal direction, and in this crude laboratory he worked alone.

But because of the sincerity and truth of the man, and the wisdom and value of his purpose, his little laboratory immediately began to grow. The writer can recall the time when any medical laboratory in the United States that wanted a pure culture of the tubercle bacillus must send to Dr. Trudeau's little laboratory to obtain it.

And so the Trudeau Laboratory, because it was good, grew until today it is housed in a beautiful fireproof building, though of modest and suitable size, and furnished with the most modern appliances, and offering to its little group of earnest workers the best of opportunities to carry on their investigations, but it is always just the child of Dr. Trudeau's brain, the logical completion of one of the projects that lay closest to his heart.

The series, of which this volume is the first, represents the results of the researches of a part of the staff of the Trudeau Sanatorium along one of its lines of thought, and we hope that it may aid the medical profession in its combat against one of mankind's bitterest and most ruthless foes, and in sending it forth we shall feel that we have to the best of our ability carried out one of the precepts of Hippocrates, the Father of Medicine, that we should "by precept, lecture and every other mode of instruction impart a knowledge of the healing art."

Walter B. James, M.D.

PREFACE.

THE frequency with which pulmonary tuberculosis is complicated by intestinal tuberculosis has been noted at autopsy for years by many pathologists. It occurred to the authors that this complication might not always be only a terminal condition; and, furthermore, that it might occur early enough in the course of the pulmonary tuberculosis to diminish greatly the chances of recovery from the lung lesion. Until they began their studies, no one was able to say when this dreaded complication first arose and, except in the terminal stages, few would venture a guess as to the presence or absence of intestinal tuberculosis in spite of the occurrence of many suggestive symptoms. The result of this unfortunate condition reacted very strikingly upon all attempts at treatment, for it is impossible, of course, to evaluate any form of treatment when the diagnosis is always in doubt. This difficulty of diagnosis without question explains why intestinal tuberculosis has for years received so little attention from students of tuberculosis. The method of diagnosis herein described has radically changed this point of view. Since the publication of their first communication a greater number of apparent recoveries from intestinal tuberculosis have been recorded than can be found in the literature up to that time. The reception accorded their work convinced them that all preceding work on the roentgen-ray diagnosis of intestinal tuberculosis had made no permanent impression whatsoever upon those interested in the study of pulmonary tuberculosis. For these reasons the authors deemed it a duty to collect in one volume such data as would enable one to diagnose or to exclude secondary intestinal tuberculosis in any stage.

The authors feel under obligations to many friends and associates of whom they can name but a small number. To Dr. Leroy U. Gardner, they desire to express their indebtedness not only for permission to publish his work but also for many suggestions and

criticisms. To Dr. Reuben J. Erickson for his splendid study of the symptoms of intestinal tuberculosis, they return thanks. To Dr. John N. Hayes for his careful blood counts they are grateful. To Dr. Edward R. Baldwin for his kindness in reading the manuscript, to Miss L. Isobel Ross, who has worked so conscientiously upon the bibliography, and to Miss Ruth Taub, their efficient secretary, they are greatly indebted. To their colleagues in Saranac Lake, who have put their cases and their records so generously at their disposal, they wish again to express their thanks.

L. B.
H. L. S.

Saranac Lake, 1926.

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INTESTINAL TUBERCULOSIS.

INTRODUCTION.

Intestinal tuberculosis is ever assuming more importance in the treatment of pulmonary tuberculosis, for it is the most frequent of all tuberculous complications, not even excepting laryngeal tuberculosis. Until recently, when the roentgen-ray diagnosis was perfected, no one has been able to state when in the course of pulmonary tuberculosis intestinal tuberculosis began, for it can exist for a long time, and cause no symptoms. If it were only a terminal complication, arising when all bodily resistance has vanished and all hope of recovery has gone, it might demand less serious attention than it does today, when we know that it complicates even some minimal or very early cases, is much more common among the moderately advanced and exceedingly frequent in the advanced and terminal stages of pulmonary tuberculosis.

Every physician, who treats pulmonary tuberculosis, has been perplexed from time to time by the frequent digestive disturbances that occur in this disease. As a matter of fact in any disease whose average duration, including all the acute cases, is two or three years and in which the average duration of the more chronic cases is five years, digestive disturbances are, as one can readily understand, very likely to occur sooner or later. The cause of this is not difficult to discover. The treatment of pulmonary tuberculosis depends on the internal or vital resources, if we may so term them. The general condition of the patient is looked upon as of paramount importance for recovery. The treatment as a rule is very passive. The patient is told to take no exercise, to be in the open, and to eat as much as possible. The result of such a method of treatment is that the only form of activity in which the patient is allowed any discretion is in eating and the natural result is that many overeat. Gastro-intestinal troubles often develop early in the disease. In fact, some occur even before the diagnosis is made and may recur from time to time until the patient is gathered to his fathers. The main question and one hitherto impossible of accurate solution is whether these gastro-intestinal disturbances are functional and due: (1) To the poisons of the tubercle bacilli; (2) to the poisons of the disease;

(3) to the poisons absorbed from the intestinal tract, if we accept for the sake of argument, the theory of auto-intoxication; or (4) finally indeed, whether they are due to organic disease in the gastro-intestinal tract, possibly tuberculous in origin.

Our studies have led us to believe that many patients, who otherwise might make a good recovery have been so seriously handicapped by an unsuspected intestinal tuberculosis that they have lost all chance for recovery before the diagnosis of this most insidious and most frequent complication was made. It was first with an idea of the selection of early cases for operative treatment that we began our studies, but later experience has shown us that other forms of treatment are more desirable in many instances.

Furthermore, some have advanced the view that tubercle bacilli, which occur almost constantly in the intestinal contents when present in the sputum, are more easily absorbed through the bowel wall when ulcerative intestinal tuberculosis is present. In fact, these observers maintain that in such cases infection of the blood and by inference reinfection of the lung is by no means uncommon under these conditions.

Such views have led us to publish this book, for the benefit, we hope, of many sufferers from pulmonary tuberculosis.

The reluctance of some to employ the methods of diagnosis described in these pages might be due, we thought, to the considerable difficulty encountered in mastering the recent anatomical and physiological data concerning the intestinal tract, facts which must be known before a diagnosis of abnormal conditions can safely be made. For these reasons, we have reviewed somewhat fully knowledge which at times may appear elementary and unnecessary in such a book.

We should like to emphasize in the beginning that deductions based upon the methods described are to be made only in connection with patients suffering from pulmonary tuberculosis. These methods can reveal, in our opinion, the existence of an ulcerative colitis, which in the presence of pulmonary tuberculosis is practically always of a tuberculous nature. That hypermotility of the colon exists only in ulcerative colitis, we would be the last to claim, but, when coupled with filling-defects, easily determined in most instances by this method, we feel that an ulcerative condition undoubtedly is present. The absence of hypermotility and of filling-defects excludes for practical purposes the presence of tuberculous colitis. Such findings do not exclude of course a healed tuberculous colitis or one occurring later.

CHAPTER I.

THE HISTORY OF INTESTINAL TUBERCULOSIS.

The history of intestinal tuberculosis may be divided into 4 periods:

I. The "Dysentery" period, during which it was noted that diarrhea frequently occurred late in the course of pulmonary tuberculosis, but little consideration was given to the fact that it might be due to the same disease in a different organ. This, the period of Clinical Observation, extended from the time of Hippocrates to that of Bayle (1810).

II. The period of "Tuberculous Diarrhea," during which the lesions in the intestine were described and noted as tuberculous and the diarrhea definitely connected with them. This period of Pathological Observation extended from the time of Bayle (1810) to the time of Louis and Rokitansky (1825–1842).

III. The period of "Pathological Study," during which the pathological changes were fully described and attempts made to connect the varying symptoms with the various changes in the lesion found at postmortem. The diagnosis during life was always uncertain. This period of Pathological Study lasted from the time of Louis and Rokitansky to 1919.

IV. The period of "Roentgenological Diagnosis," during which the diagnosis and consequently the scientific study and scientific treatment of intestinal tuberculosis could be accurately pursued, dated from the time the roentgenological diagnosis of intestinal tuberculosis was placed on a firm foundation in 1919 by the work of the authors at the Trudeau Sanatorium.

THE "DYSENTERY" PERIOD.

The occurrence of diarrhea in the later stages of pulmonary tuberculosis was noted by Hippocrates and commented upon by Galen, Celsus, Aretæus, and others. In his aphorisms Hippocrates wrote: "Phthisical persons, the hairs of whose head fall off, die if diarrhea sets in;" and again, "Diarrhea attacking a person with phthisis is a mortal symptom." In speaking of phthisis, he noted that the bowels were variously constipated and again immediately

in a loose state, but toward the termination in all cases a violent looseness of the bowels occurred. All ulcers described by Hippocrates were classed under dysentery, a method followed by Celsus and Aretæus.

Little of importance was noted about intestinal tuberculosis until the Seventeenth Century. The chaotic state of knowledge during this period may be gained from a few quotations.

Thomas Sydenham (1634–1689), discussing pulmonary tuberculosis, wrote: "Lastly to close the scene a looseness succeeds, occasioned partly by corrupt humors, discharged from the mesenteric arteries into the intestines and partly by the weakened tone of the viscera; and thus the patient perishes at length the following summer by a distemper occasioned by the foregoing winter."

The state of medicine in 1694 and its theories about intestinal tuberculosis are curiously shown in Richard Morton's Phthisiologia where under the head of colliquation he groups bronchorrhea, sweats and severe diarrhea, "due to an excretion of some colliquative matter from the blood or glands. When it takes place into the bowels a violent looseness arises, that is no less colliquative than the sweats themselves; yea and sometimes even a bloody flux (if the colliquated humor be very sharp) attended with dreadful convulsions and torments of the bowels, which bids defiance to all the medicines that are used to cure a dysentery; as I found in the deplorable case of Mr. L. Lechmere and likewise some other persons that have been in consumption, who being by that symptom thrown into the last degree of a consumption, with a Hippocratic face, died as quickly as they did miserably." "And therefore a looseness is justly to be reckoned for the most part, one of the last and fatal diagnostic signs of a confirmed consumption."

In 1700, Manget, an industrious collaborator, who edited and republished many works of the Seventeenth Century, recorded, in a revised edition of Bonnet's works, a case of a young man who died from phthisis. At autopsy he found "hailstones" (probably tubercles) in lungs, liver, spleen, mesenteric glands and intestines. He likened them to millet seed.

Early in the Eighteenth Century (1715), Johann Conrad Brunner, a Swiss physician and pathological anatomist, described a case of intestinal tuberculosis in which he found 60 ulcers (probably tuberculous) of different sizes throughout the intestines, many of them in Peyer's patches, in a patient dead of pulmonary tuberculosis.

In 1731, stenosis, probably due to intestinal tuberculosis, was described by Walther and Krisch of Leipzig.

"The third and last act of this domestic tragedy" (pulmonary tuberculosis), wrote Thomas Reid in 1785, "commences by the appearance of a purging. Though this very generally takes place toward the end of the disease, when it terminates fatally, yet sometimes there is scarcely any appearance of it. . . From having been costive during the former period of the disease, the patient has now frequent motions in the day, which soon become a confirmed diarrhea; everything taken into the stomach quickly running off by the bowels."

THE PERIOD OF TUBERCULOUS DIARRHEA.

G. L. Bayle, "the founder of correct teaching about tuberculosis," wrote in his Recherches sur la phthisie pulmonaire, published in 1810, as follows:

"In some remarks upon tubercles published some years ago[1] I have described the lesion of the tubercle in cervical and mesenteric glands and I have spoken in detail of the mucous membrane of the intestine affected with the same disease. Of all the changes which occupy us, there are perhaps none more remarkable than this ulceration, on account of the diarrhea, which it frequently causes. We will speak of the symptoms in the article on treatment but we place here a proportional table of 100 phthisical cases. One can judge from this table of the frequency of these ulcerations. Of 100 phthisis cases dying in the year xii, 33 had a healthy alimentary canal and 67 presented ulcerations in the intestinal canal.

"In some of the patients whose intestines were ulcerated the intestinal ulcer was the result of transparent miliary granulations, but ordinarily miliary tubercles causes this affection." He recognized also that diarrhea could occur in pulmonary tuberculosis in the absence of intestinal tuberculosis.

This is one of the earliest clear-cut statements connecting the ulceration in the intestine with the tuberculous disease of the lung and with the frequent concomitant diarrhea.

Bayle's publication was followed in a few years (1819) by that of his friend, R. T. H. Laënnec, one of the great figures in tuberculosis history. In his book on auscultation (1819), Laënnec called attention to the fact that the lymphatic tissue of the intestine can soften without ulceration of the mucous membrane, but that such ulceration is much more common in tuberculosis. He described the tuber-

[1] Jour. de méd., chir. et pharm., germinal an **12** (1803), tome 6, p. 3; et germinal an **13**, tome 10, p. 42.

cles in the submucosa and under the peritoneum and found them most often in the terminal ileum. The ulcers can penetrate to the peritoneum but perforation is uncommon. If it occurs into the peritoneum, acute peritonitis with tympanitis occurs but more usually it opens into a walled-off area, often lined with many tubercles and without any symptoms. The mucous membrane is usually pale. Laënnec thus agreed in general with his friend Bayle. He spoke of colliquative diarrhea as a companion of pulmonary tuberculosis. Tubercles, he thought, rarely occurred in the lung alone.

THE PERIOD OF PATHOLOGICAL STUDY.

This period was inaugurated in 1825 with the publication of the anatomical pathological studies of P. C. A. Louis (1787–1880), the originator of the statistical method in medical investigation. His observations are minute, accurate and painstaking. They are so valuable that a rather full abstract has been included in this chapter. They herald the beginning of a new era. Taken together with the clear and valuable contribution of Rokitansky, they have formed the basis for all later contributions to the study of intestinal tuberculosis. During this period many other pathologists also took up the study of intestinal tuberculosis.

Louis, in 1825, published his pathological studies on phthisis, which contained many interesting facts concerning intestinal tuberculosis. In 60 cases, he found 3 with duodenal ulceration. In the small bowel he noted in the submucosa "granulations," resembling tuberculous tissue or at times harder and firmer, semicartilaginous, averaging about the size of a pea. They varied in number but were usually more numerous nearer the cecum. When small, the overlying mucous membrane was normal, when larger, it was generally more or less red, thickened, softened or even destroyed. The "granulations" then began to decrease in size, leaving an ulceration with white indurated edges. He further described tuberculous "granulations," equally numerous with the former type and situated around the granulations or in the other layers of the bowel wall. They were numerous near the cecum. Small ulcerations succeeded these granulations and were produced in the same manner as tuberculous cavities in the lungs. Such granulations existed in 36 out of 95 cases.

Ulcerations were more numerous, occurring in 78 instances or nearly five-sixths of the cases. The number, depth and dimensions of the ulcerations increased as the cecum was approached. The

lower third of the ileum was the portion usually involved and in only 3 instances were they confined to the middle third. They began opposite the mesentery. When small they were round, when larger, elliptical, assuming the shape of the Peyer's patches, rarely linear. Their color was whitish when small, often gray or mingled with red when larger, and occasionally blackish or reddish-brown when linear in shape. When small the denuded submucosa was slightly thickened, smooth and the muscularis apparently normal.

The larger ulcers had an irregular surface with bits of mucous membrane remaining like islands. In others the muscularis was exposed, more or less thickened, rough, grayish or whitish, and presenting at times tuberculous granulations. In fact as any of the various coats became denuded, it first thickened somewhat before ulcerating. The larger ulcerations were frequently the result of the fusion of smaller ulcers.

Similar lesions with the exception of the semicartilaginous granulations existed in the large bowel. In 27 of 95 cases the whole of the mucous membrane was red. There was some softening, (65 cases) and thickening which is usually associated with ulceration.

Ulceration was frequent (70 cases) and in 13 cases had tuberculous granulations around it. Ulcerations were found in the cecum in 34, in the ascending colon in 37, in the transverse in 25, in the descending colon in 8 and in the rectum in 32. The bases may be dark or pink and discovered then only after washing the bowel. The shape is usually elongated and they may be placed in any direction. The largest ulcers, usually irregular and grayish, are found chiefly in the cecum and ascending colon and in the ascending colon may encircle the gut or be 8 or 9 inches long, inclosing islands of normal mucous membrane.

One-fourth of his 123 cases had diarrhea from twenty to five days before death. The stools were usually infrequent and in a few colic varying in intensity was present. Ulcerations were present in one-half the cases, in either the small or large bowel, sometimes in both, which, with one exception in the small and two in the large, were small and few in number.

"There was an exact correspondence between the symptoms and the alterations to which they might be attributed. For if the diarrhea had preceded death only by a few days, this seemed to be equally the case with the ulcerations and softening of the mucous membrane of the colon." He considered such ulcers to be very recent. The diarrhea was less copious in patients in whom only ulceration and no softening of the mucous membrane had occurred.

The diarrhea of long duration was either continued or remittent. In 15 patients with remittent diarrhea, with periods of remission of eight to twenty days, the stools were generally scanty, the colicky pains rare, though the duration of the trouble had extended from forty-eight days to fifteen months. Ulceration was present in the small bowel in 10, in the large in 6, and, with two exceptions for both bowels, the ulcers were small. Softening of the mucous membrane was present in 10 and in 3 was red and thick. It is of interest to note that these 15 patients presented nearly the same pathological changes, both as to intensity and extent, as those in whom the diarrhea commenced, as it were, only a few days before death. It would seem natural then to conclude that the lesions observed at autopsy were of very secondary import in the production of the diarrhea; that they began in both series toward the close of life.

Chronic and continued diarrhea was present in 41 patients, 35 of whom had ulcerations in the small and 31 in the large intestine. In 12 cases the ulcerations extended throughout the small bowel and were of considerable size, often an inch in diameter, whether generally or locally distributed. Extensive ulceration of the large bowel was present in 19, and 30 had softening of the mucous membrane, in 17 of which it was red. The diarrhea in these patients was more or less violent, and accompanied by colicky pain. One case of five months' duration had 12 to 15 stools a day, often copious. Thus after long continued diarrhea, extensive and numerous ulcerations were present, but they were more intense and evidently of longer duration than in the cases with remittent diarrhea.

Large ulcerations were found in both intestines in 6 patients in whom diarrhea had been copious and continued for two to eleven months. If confined to the small bowel, the diarrhea was no less chronic and continuous. However, the change in the large bowel was the chief source of such diarrhea. To be able to predict with some certainty the existence of large and numerous ulcerations, it is necessary not only that the diarrhea should have been chronic and continuous, but that the stools should have been frequent. No exception to this rule came under his observations. If the stools have a very foul odor, it makes such a diagnosis more certain.

Among the phthisical patients who died in the wards of M. Lerminier during the last six years, writes G. Andral in 1843, only one-fifth had a normal intestine. The first change noted is an alteration of the color of the mucous membrane, which becomes more or less bright red, brown, grayish, slate-colored, or studded with

small black elongated points. The second change is softening which results merely in liquid pulp.

"The opinion which consists in considering intestinal tubercles as having their site in the follicles, has been already published by several authors, and M. Billard[1] has recently supported it by new facts.

"In intestines full of tubercles, we have often met ulcerations which bore a great resemblance to pulmonary caverns; they represented, as the latter, anfractuous cavities, separated by bands of an irregular form."

He has noted more than once vegetations, fungous growths, variable in form and in size, projecting from the inner surface of the intestinal mucous membrane.

Twice he has seen very extensive gangrene of the mucous membrane which was limited to this coat. Tubercles arise under the mucous membrane. Nothing is more common than the existence of ulceration toward the end of the small intestine and in the cecum of phthisical patients with chronic diarrhea.

The intestinal irritation may decrease the cough or dyspnea, acting like a revulsive. He notes that even in chronic or obstinate diarrhea, there may be only reddening of the intestinal mucous membrane.

"The symptoms are very different when the inflammation assumes a chronic character; it is then reduced merely to its local symptoms, and these again are often but slightly marked. Thus, for instance, many phthisical patients, whose intestinal canal is found inflamed, ulcerated, filled with tubercles, and very much disorganized over a vast extent, have not complained of any considerable pain, affecting the abdomen; this is the most general case. The belly remains soft, nor does pressure on it occasion pain. In some each alvine evacuation is preceded only by a slight colic; in others the stools are not even preceded by any painful sensation. The evacuations most commonly consist either of a clear, serous liquid, which the patients compare to water, colored yellow or green, or of thready, commonly called glairy mucus, or of a grayish, very fetid, paplike substance, or finally, though more rarely, of real pus. It seems to us impossible to connect each of these species of evacuation with a determinate alteration of the intestine; all they indicate is, that this intestine is altered; and as, 9 times out of 10, this alteration is the result of inflammation, we must thence conclude that the diarrhea in phthisical patients is, in this same proportion, the

[1] De la membrane muqueuse gastro-intestinale dans l'état sain et dans l'état inflammatoire, 1825.

sign of an enteritis or a colitis, and most frequently of one or other of these inflammations combined. The long duration of the purging is a probability for thinking that the intestines are ulcerated; but in this respect there never is any certainty. There is one thing which always appeared to us very remarkable, namely, that in some phthisical patients, who had slight purging, and that for only a little time, without any other symptom affecting the abdomen, we have found numerous ulcerations either in the small intestine, or in the cecum, while in others, whose diarrhea was of a longer standing and more profuse, we sometimes found nothing but mere injection of the mucous membrane.

"It is not always at the same period of pulmonary phthisis that the intestinal canal becomes affected. In this respect phthisical patients may be divided into four classes; in the first we shall place those in whom the affection of the intestine seems to precede that of the lung. In the second we shall range those in whom this double affection arises and proceeds simultaneously. The third class will comprise those patients, who for a long time presented only some signs of pulmonary tubercles, and in whom the purging or other signs of intestinal affection supervene only at an advanced period of the pulmonary phthisis. The fourth class shall be reserved for those who die phthisical, the intestinal canal remaining uniformly healthy, which is the most uncommon case. We shall present a summary of our observations on these different cases.

"We have often seen persons at La Charité, who were for a long time laboring under chronic diarrhea when we first saw them. They were already in a more or less advanced stage of marasmus; they had a continued febrile disturbance, with an accession once or twice a day; they had no cough, nor had they ever had any; their breathing was free, auscultation and percussion announced a healthy state of the pulmonary parenchyma. They had, if I may so say, merely intestinal phthisis. But after a longer or shorter time, a little cough began to appear, deep inspirations became impossible, more or less profuse hemoptysis took place; auscultation and percussion as yet gave no information, but the symptoms altogether were sufficient to incline us to suspect the existence of pulmonary tubercles, which being developed consecutively to the intestinal phthisis, became an alarming complication of it; if then, the individual died, we found traces of chronic inflammation in the intestine, while in the lung there was as yet nothing but crude tubercles, and those few in number. At other times, the patients prolonged their existence for a longer time; the pulmonary tubercles went on increasing,

and they did not die until the pulmonary parenchyma had numerous caverns, the presence of which was detected by auscultation."

Pulmonary tuberculosis may precede the chronic enteritis, but often remains stationary while the enteritis advances. The symptoms may arise simultaneously, advance together or frequently the bronchitis and enteritis may alternate, but finally coexist.

Corbin, a French surgeon (1830–1831), wrote on the importance of tuberculous intestinal stenosis and perforation, and showed the connection of stenosis with the healing process, which, with Andral, he called scirrhus degeneration. Blatin (1868) and Duguet (1877) described this same condition limited to the cecum, and a little later the surgeons began to operate for the tuberculous cecal tumor due to the hypertrophic form of cecal tuberculosis.[1] Hartmann and Pilliet (1890), whose contribution placed this type of tuberculosis on a scientific basis, and Conrath, whose excellent work was published in 1898, have added greatly to the subject. Those interested in this type should consult also the articles of Lartigau, Brunner, Erdman.

In his illustrations of Pulmonary Consumption (1837), Samuel G. Morton, of Philadelphia, a pupil of Laënnec, calls attention to the fact that diarrhea may commence early in pulmonary tuberculosis and continue to the end. Neglect of this symptom involves fatal consequences. The usual cause, he continues, is ulceration of the intestines, most often in the ileo-cecal region. Its occurrence may suspend or diminish cough, but the one is no sooner arrested than the other returns. Connected with the diarrhea is a very irritable stomach which refuses all food.

Perforation of tuberculous ulcers was described by Laënnec and later by Voisin (1833) and Dupan (1835). Andral stated that perforation could occur from the peritoneal side, which was confirmed by Piégu (1840).

Stenosis was described, as stated, as early as 1731, but Ossipowski (1840) attributed the stenosis to scars, brought about as a result of healing, forming a band or valve. Above the stenosis hypertrophy of the muscle and dilatation of the bowel occurred. Tardieu, under whom Ossipowski worked, confirmed his pupil's observations. Koenig (1892), Bernay (1899), Patel (1901) and Crowder (1902) have made interesting contributions to this subject.

In 1841, Bodart described tuberculosis of the cecum or ileo-cecum as a special form of the disease, which others have done more recently. Leudet in 1859 described tuberculosis of the appendix.

[1] Gussenbauer (1882), Czerny (1886–1887) Suchier, Bouley (1887).

In 1842, W. W. Gerhard, of Philadelphia, stated that the tuberculous disease of the follicles of the intestines is very intermittent at first and the symptoms vary incessantly, diarrhea often occurring for several days and then being followed by constipation. After a certain time the diarrhea may entirely cease and the follicles, which are the seat of the deposition, will cicatrize.

In 1845 Rokitansky published his classical description of intestinal tuberculosis, which has served as the foundation of all later articles. We quote his section on tubercle from his work on pathology.

"The presence of tubercle," he wrote, "in the tissue of the intestinal mucous membrane, and by extension, in the deeper-seated coats, constitutes a most important disease—tuberculosis of the intestine in the broad, tuberculosis of the intestinal mucous membrane in the narrower sense. It may proceed to ulcerative destruction, and thus establish genuine intestinal phthisis.

"With us this affection rarely occurs in the idiopathic form, except during the first years of life. It is commonly the consequence of pulmonary tuberculosis, and in the majority of cases, takes place after the latter has attained the suppurative stage (pulmonary phthisis), and the general tuberculous cachexia has become fully developed.

"The course it runs is frequently chronic, but much oftener acute; the latter is more particularly the case when it follows the tumultuous fusion of numerous pulmonary tubercles. The tuberculous deposit offers corresponding varieties in reference to its original form, its seat, and its metamorphosis.

"In the chronic affection we find the mucous membrane, and the adjacent layer of submucous cellular tissue, to be the original seat of the tuberculous deposit; there is no perceptible inflammatory action, and the disease appears in the shape of gray, transparent, tuberculous granulation, which softens at its center, and is gradually converted from within outward into the yellow cheesy tubercle. It seems blended with the mucous membrane, and projects into the intestinal cavity in the shape of a sessile, hard nodule.

"When the local appearance of tubercle takes place in the acute form, there is considerable inflammatory action. The deposit is affected similarly to that occurring in the pulmonary cells; in the first instance it is deposited in the cavity of Peyer's glands, then in the solitary follicles, and last, in every other part of the intestinal mucous tissue; it appears in large masses, and in the shape of yellow, cheesy matter, which speedily undergoes a purulent transformation. The surrounding tissue is found extensively congested, reddened

and turgid; and when the deposit is excessive, the mucous membrane of an entire coil may be in a state of congestion and irritation. In this case tuberculous tumors, either scattered over the surface of the intestine or more or less accumulated, are found occupying Peyer's patches, offering considerable projections and distinguishable through the mucous membrane by their yellow tinge.

"Tuberculous deposit in the intestinal mucous membrane, being the result of a fully developed tuberculous cachexia, commonly advances rapidly to softening, and this process is affected with peculiar violence in the second variety. The investing mucous membrane gives way at its most elevated point, and as the orifice enlarges, the suppurating tuberculous matter escapes.

"A cup-shaped ulcer, of the size of a millet seed or a pea (the primary tuberculous ulcer) results; its margin is firmly attached, rounded and indurated, and of a pale or red color in proportion to the reaction that occurs in the surrounding tissue; its base is either formed by the condensed submucous cellular layer, or by the granulated texture of the parietes of the dilated follicle. It is only in very rare cases that the tubercle fuses under the mucous membrane without giving rise to perforation; it then forms an inclosed abscess, which enlarges at the expense of the submucous cellular tissue (vomica submucosa).

"The ulcer increases in size with more or less rapidity, loses its original form, but only to exchange it for a more characteristic secondary one.

"The increase is effected by fusion of the tuberculous infiltration of the margin of the ulcer, and by concurrent suppuration of the tissue. In the first instance, the small adjoining ulcers coalesce into one of larger size; the common base presents sinuous projections of the common margin of mucous tissue, ridges of mucous membrane may be seen traversing it in various directions, or even solitary insular remnants of this tissue are found upon it.

"If this process has occurred, as it does in acute intestinal tuberculosis, in one of Peyer's patches, the ulcer may, on account of the elliptic form prescribed by the shape of the glandular apparatus, be mistaken for a typhoid ulcer, but we shall immediately point out that the peculiar relations of the margin and the base afford a satisfactory clue to the diagnosis.

"The ulcer, which is formed by a coalition of other smaller ulcers, enlarges in the same manner as the original solitary ulcer in the direction of the intestinal circumference, and at last presents a zone of varying width and uniformity. Its margin is sinuous or dentated,

inverted and tumid, and is formed by mucous membrane of a light red color; from the latter being infiltrated with a transparent gelatinous substance, an analogy is offered with the gelatinous infiltration occurring in the vicinity of tuberculous pulmonary abscesses. The base is formed by callous cellular tissue of a dirty white color, underneath which the remaining intestinal layers are found similarly condensed and tumefied.

"Both in the marginal tissue and at the base we find a deposition of gray, or more commonly of soft, yellow, tuberculous matter. The ulcer presents a very peculiar appearance, on account of the remnants of mucous membrane seen on its base. These adopt the characters of the margin, and become infiltrated with gelatinous matter, so as to form crispated, transparent, condylomatous excrescences of a light red color.

"In the same manner as the tuberculous ulcer extends laterally, it may advance in the opposite direction, and thus giving rise to perforation, cause sudden death. Secondary deposition of tuberculous matter may equally take place in the callous cellular tissue of the base, and as it fuses at this point, in the muscular and subserous layers also. The peritoneum may become perforated in consequence of tuberculous suppuration being established in it, or in consequence of mortification induced by the approach of an abscess. It follows that the tuberculous ulcer perforated the intestinal parietes without losing its original character, inasmuch as the progress of the tuberculous affection is not arrested by an isolating tissue; in this it differs from the typhoid ulcer, which does not perforate the intestine in its original form, but affects the part beyond the submucous cellular tissue in its degenerated character.

"At an earlier or later period we find moderate inflammation attacking points of the peritoneum which correspond in position to the intestinal ulcer; a fibrinous exudation results, which is entirely, or in part, converted into tubercle; in the latter case it is partly converted into cellular tissue. By the intervention of this new product an adhesion is often effected at the point of ulceration, between the intestine and a neighboring organ, *e. g.*, the bladder, the omentum, and thus a more or less substantial impediment is offered to the free discharge of the intestinal contents into the peritoneal cavity on the occurrence of perforation.

"The mesenteric glands, lying in the vicinity of the affected portion of the intestine, are variously enlarged; in the primary intestinal tuberculosis of children they frequently attain the size of a walnut or hen's egg; they appear tuberculated and pale, and present

a deposition of grayish, medullary, and hard, or of yellow, grumous, and deliquescent, tuberculous matter.

"The small intestine is the common seat of intestinal tuberculosis, and in most cases the disease is limited to this part, still it often passes on to the colon and descends to the rectum, or it ascends into the jejunum, and in very rare cases mounts to the duodenum and the stomach. Sometimes it is much advanced in the colon, and then appears to have been first developed at this point and subsequently to have extended to the small intestine.

"We may gather from the circumstances accompanying intestinal tuberculosis, that the further it has advanced the less a cure is to be hoped for. Still in the same manner as in the tuberculous abscesses of the lungs, we sometimes observe a healing process established in a few among a large number of ulcers. It takes place in the following manner:

"The first indispensable condition is the cessation of all secondary tuberculous infiltration at the margin or base of the ulcer; the callous base is then condensed into a fibro-medullary cord, and the edges of the ulcer approach one another. This process sometimes advances so far, that the dentated edges almost touch, and between them a whitish, callous cord may be observed. Occasionally, the edges are soldered together over the callosity, yet so as to have a fissure at one end of the ulcer. In very rare cases an entire consolidation is effected.

"In consequence of the contraction of the ulcer, a cicatrix forms on the surface of the intestine, which presents a more or less elevated tumid ridge on the internal surface of the intestine. If the ulcer was of considerable size, or if it encircled the entire intestine, a callous annular ridge remains, which diminishes the caliber of the intestine, and when viewed from without, occasionally gives rise to an appearance of invagination.

"Thus the cure of a tuberculous intestinal ulcer is always accompanied by a diminution of the intestinal caliber."

Spillmann, Rindfleisch, Girode, Cornil and many others, writing upon the finer anatomy of intestinal tuberculosis, have acknowledged their indebtedness to Rokitansky.

Lebert in 1849 noted that the lymph vessels about the ulcers were at times filled with tuberculous masses. He thought the submucous and muscular coats offered some resistance to the ulceration.

Edwin Klebs, first to pass from culture to culture the tubercle bacillus and always an upholder of the infection theory of tuberculo-

sis, was among the first to undertake systematic feeding experiments (1868). His graduation thesis (1858) dealt with tuberculosis and the intestinal tract, and he later emphasized the danger of milk from tuberculous cows for small children. In 1868, by feeding tuberculous masses from cattle and men, he produced tuberculous ulcers in guinea-pigs, fully as large as those in men, and later tuberculosis of the lymph vessels, of the serous coat of the intestines, and of the mesenteric glands. He considered the intestine to be one of the chief seats of the organism producing tuberculosis, and was the first to advance the view that intestinal tuberculosis in man was caused by the specific germ in the swallowed sputum.

In 1862, J. A. Villemin described calcified tubercles in the intestinal walls. They may be found under the peritoneal coat of the small bowel.

A brief résumé of experimental intestinal tuberculosis is given in the section devoted to that subject (see Chapter X).

"At the lower part of the ileum in spaces occupied by the Peyerian and solitary glands," writes Austin Flint in 1875, "numerous round bodies of the size of small peas projected into the canal. Some of these contained a liquid like softened tubercle; others were white and cheesy. There was no thickening nor any appearance of inflammation around these bodies. They were most abundant near the cecum. It is certain that the amount of ulceration cannot be accurately estimated by the prominence of the diarrhea.

"There are no facts which show that recovery ever takes place in cases of phthisis when the prominence and persistence of diarrhea warrant the conclusion that intestinal ulceration exists. The cessation of the diarrhea is not proof that the ulcers have healed. In a study of 31 cases 29 ended fatally, the shortest in three months, the average under thirteen months, showing that diarrhea has a notable influence in shortening the duration of phthisis."

In 1 case the stools contained liquid fat in considerable abundance.

"The conclusions drawn from the analysis of 31 cases with regard to diarrhea are as follows:

1. "Diarrhea, when prominent and persistent in phthisis, denotes, as a rule, ulceration in the small intestine, with enlargement of the mesenteric glands.

2. "The prominence of this symptom is not always a criterion of the amount of ulceration.

3. "Perforation of intestine is an occasional accident in connection with intestinal ulceration.

4. "Cases of phthisis in which diarrhea represents intestinal

ulceration, offer very little, if any, ground for the hope of recovery; it is doubtful if cicatrization of the ulcers ever takes place.

5. "Diarrhea in some cases ceases for a time, notwithstanding the existence of ulceration.

6. "The average duration of phthisis with diarrhea is considerably shorter than the average duration of the disease in an indiscriminate collection of cases.

7. "In some cases characterized by the prominence and persistence of diarrhea, death takes place with a very small amount of pulmonary disease, and, under these circumstances, death may take place without the formation of cavity in the lungs.

8. "Hemorrhage incident to intestinal ulceration may occur, and the loss of blood may be the immediate cause of death."

THE PERIOD OF ROENTGENOLOGICAL DIAGNOSIS AND STUDY.

This period begins in 1919 with the publication of the first article by the authors. That roentgen-rays had been used previously to their work is mentioned in their various articles, but the work was, with a few overlooked exceptions, unconvincing, meager, and itself attracting no attention, proved of little help in the treatment of pulmonary tuberculosis, where intestinal tuberculosis plays such an important part. In 1919, the attitude of clinicians interested in pulmonary tuberculosis was that it was impossible to diagnose intestinal tuberculosis in its early stages and often some doubt was expressed in an individual case even when the intestinal disease seemed to be well advanced. In fact the attitude of Joseph Walsh, who wrote in 1909, was that as it was impossible to diagnose intestinal tuberculosis with any degree of certainty, and as such a diagnosis caused patient, physician and nurse to abandon the strict treatment which might lead to recovery, no diagnosis of intestinal tuberculosis should be made, was also the attitude of many other workers in tuberculosis, who considered intestinal tuberculosis as always a fatal complication and, if perchance, a patient suspected of having it recovered, the diagnosis was considered to be in error. There were not lacking among these workers many men, who frankly averred that it was impossible either to affirm or to exclude the presence of intestinal tuberculosis in any individual case.

Such then was the condition of the diagnosis of intestinal tuberculosis when the authors began their work. They were aware of the fact that Prof. E. W. Archibald, of Montreal, had suggested to

Dr. A. H. Pirie that he examine with roentgen-rays the patients with intestinal tuberculosis referred to Archibald for operation.

As a result of his studies, Pirie published a note in Archibald's excellent paper, which appeared in 1917 and follows in full:

"Dr. Archibald has asked me if I could write from memory a summary of my findings in tuberculous conditions of the intestine. I do so with pleasure, as it was the most interesting work I did for about a year before the war began. Writing as I do without notes from a base hospital in France, I well remember the disappointment we had, as in several successive cases of tuberculous cecum I failed absolutely to help in the diagnosis. Some bad luck seemed to dog my steps, for whereas we had formerly been able to show by means of a barium meal the position, shape and size, of the normal cecum, yet we failed, in these tuberculous cases, to catch the cecum at the proper time when it was filled by the meal. Finally, the cause of our failure struck us. We could not show the cecum filled by a barium meal because it never did fill when tuberculous ulceration existed. Setting out with this as a working hypothesis, we examined the cases at half an hour intervals from four to twelve hours after the barium meal. We found our theory correct in each case of tuberculous cecum, viz., the cecum never filled up with the meal. Each small squirt of barium that left the ileum was quickly passed on past the cecum and collected elsewhere in the large colon, either in the transverse part or the descending. So that our conclusion was, that in a tuberculous subject with symptoms suggesting tuberculous cecum, if the cecum did not fill from four to twelve hours after the barium meal when examined at intervals of about half an hour, then this want of filling confirmed the diagnosis. On the other hand when it did fill as it does in a normal individual, it negatives the diagnosis of tuberculous cecum. We had this latter experience confirmed in 1 case where a clinical diagnosis of tuberculous cecum had been suggested; the roentgen-ray showed a cecum filled by barium, and at operation a non-tuberculous cecum was found."

It is of interest to record that the significance of Pirie's note entirely escaped the attention of the authors and of all workers, who passed it by apparently without a second thought. The authors approached the problem from an entirely different angle and were first impressed with the hypermotility, as evidenced by the complete or nearly complete evacuation of the barium meal in twenty-four hours or less. As they progressed in their study they found that their observations agreed in part with those of Pirie.

After the authors had worked out their method of diagnosis and began to search the literature for evidence corroborating their work, they discovered that, in 1911, Stierlin published an excellent article in which he described 6 cases of cecal tuberculosis and 1 of non-tuberculous colitis, in all of which he made a diagnosis of ulcerative colitis, confirmed at operation by Wilms and based upon inability to visualize the barium meal in the cecum at certain times when it should normally have been present. The picture suggested hypermotility and upon this fact Stierlin diagnosed ulceration of the large bowel at the site which threw little or no shadow. He referred to the fact that the partially filled portion shows in some cases fine marbling or long drawn out shadows, an irregular contour, that haustral segmentation is often absent, and a rigid cylindrical form may be present. He failed to emphasize the importance of hypermotility and of filling-defects that departed from normal in any way, and interpreted some of the "marbling" as due to ulceration. He suggested the use of the opaque enema in difficult cases.

"In infiltrating and ulcerative involvement of the cecum and ascending colon," writes Stierlin, "there is absence of the normal shadow of this part of the colon, as shown in the skiagram six or seven hours after the barium meal, while the terminal ileum and transverse colon are filled. Consequently the early as well as the later stages of the so-called cecal tuberculosis can be diagnosed by this means even in cases undiagnosable by the ordinary clinical methods" (see also p. 159).

Case, in a study of the ileo-cecal region and appendix, published in 1912, stated that "ileo-cecal tuberculosis is a condition which in a certain number of cases can be almost surely diagnosed by the roentgen-ray examination," but gives no further data.

In a long article upon the diagnosis of disease of the gastro-intestinal tract by means of the roentgen-rays, Béclère and Mériel, in 1912, devoted a short notice to Stierlin's work. They say his work concerns not only the cecum but the whole of the large bowel, in which they err. They further say the ulcerated area is always empty, which is again a mistake, as it is frequently partially filled. The cause of this appearance is not unique, they continue, because sometimes it is cancerous ulceration with diffuse infiltration of the wall which is transformed into a rigid tube: sometimes it is ulceration, most often tuberculous, which affects only the superficial part without diminishing the muscular contractility. For these two sorts of lesions, so different, the roentgen-ray picture is the same. In the first the contents are hurried too rapidly without arrest, through

the ring or through the rigid tube, as in certain scirrhus cancers of the stomach. In the second case, the ulcerated mucous membrane, abnormally irritated, shows its irritation by an exaggeration of the muscular peristalsis and an abnormally rapid progression of the intestinal contents. They quote Stierlin and give no data whatsoever of their own.

Mauclaire, in 1912, in a good review of the surgical treatment of abdominal tuberculosis mentions Stierlin's work and reproduces the plate of 1 case.

Schwarz and Novascinsky (1912) have also in part verified Stierlin's work. They describe the outline as being very irregular, distended with gas only at the flexures and state that the opaque column is contracted without haustration. They do not regard the streaky or marbled appearance as due necessarily to ulceration, but in general fail to emphasize the real essentials.

Kienböck, in 1913, mentions that Stierlin was the first to demonstrate the possibility of roentgen-ray diagnosis of ulceration of the large intestine and mentions the use of the opaque meal or enema. He reports 2 cases and described 2 types. The descriptions are not clear and his work apparently failed to convince other workers. He, too, failed to emphasize the essentials.

Bandelier and Roepke, in 1913, in their very full and suggestive work on tuberculosis, mention Stierlin's work in passing, as one of us mentioned artificial pneumothorax in 1907.

Schwarz, in 1914, emphasized the fact that the barium must have reached and have gone beyond the affected portion, and yet sufficient remained behind, before a diagnosis of filling-defects could be made. He mentions 1 case of hypertrophic tuberculosis and one with infiltrated walls, both positive by roentgen-ray.

Faulhaber, in 1916–1917, in a most suggestive article on tuberculosis and carcinomatous infiltration of the cecum, refers to 2 of his cases, previously reported, that upheld Stierlin.

In this article he says that a classical picture with Stierlin's sign may fail in many cases of tuberculous cecum. He thinks little of marmorization but correctly stresses for the first time the pathological smallness of the shadow of the ascending colon, its abnormal form and contour, the inelasticity of its walls, and its quickened emptying. He calls attention to the fact that, if it is narrowed and with abnormal outlines, the diagnosis is sure. He also stresses an indistinct outline. He quotes Groedel as not agreeing fully with Stierlin.

In 1917, L. Fraenkel and P. Eisen in an article on pulmonary tuberculosis, spoke of Stierlin's work and advocated the use of

stereoscopic plates in the study of intestinal ulceration. They refer chiefly to hyperplastic intestinal tuberculosis and state that, when the clinician has evidence only of constipation, the roentgenologist has already definite proof of a beginning diarrhea. Their article is of little aid in roentgen-ray diagnosis of secondary intestinal tuberculosis.

In 1918 Vidor Révész reported 3 cases, 2 of which on plates appeared positive but none came to operation or autopsy. He further states that he got many negative results and quotes Faulhaber as having the same experience. He apparently thinks little of Stierlin's work, an opinion, which seems widespread in Germany and elsewhere.

François, in 1922, reported 2 cases of ileo-cecal tuberculosis, which he diagnosed by the meal, following Stierlin's technic. He emphasized again the importance of an empty cecum while both the terminal ileum and the ascending colon were well filled.

Sigmund, in 1924, reported from Prague 8 operative cases in which the ceco-colon was empty while the ileum and transverse colon were filled (Stierlin's sign). The phenomenon was absent when the entire wall was infiltrated. It is not, he believes, a reliable sign of ileo-cecal tuberculosis.

Notwithstanding that these scattered, insufficient, and at times confusing, and even misleading suggestions had been made following Stierlin's and Faulhaber's contribution, no gastro-enterologist or student of tuberculosis appeared willing to advance the positive view that intestinal tuberculosis could be diagnosed in its early stages by roentgen-rays until the authors' work appeared. As an example, Carman, of the Mayo Clinic, in the first edition of his book on the roentgen-ray diagnosis of diseases of the alimentary canal, published in 1917, devotes a brief chapter to tuberculosis of the colon. He mentions 4 cases and states that "filling defects or obstruction with a corresponding palpable mass constituted the chief findings." In his second revised edition of 1920, published the year after our work, the whole subject is entirely rewritten and the method worked out by the authors is given in more or less detail. It is of interest to note that Carman, who rarely uses the barium meal, finds intestinal tuberculosis more frequently in the small bowel. His technic of using the barium enema alone will lead, as our experience has shown us, to some regrettable mistakes and should not be followed.

Lemon mentions that from a roentgen-ray study of 34 cases of his series of 50 cases of intestinal tuberculosis at the Mayo Clinic, a positive roentgen-ray diagnosis was made in 28. The diagnosis

was verified by operation or autopsy. It is interesting to note the failure of diagnosis by roentgen-rays in 6 of this small series of cases especially when Carman's technic is considered, and when it is compared with our results as checked by operation and autopsy (see Chapter XXIII).

In an article by Johannes Mueller published in the third edition of the *Handbuch der Tuberkulose*, edited by Brauer, Schröder and Blumenfeld and published in 1923, he states "that the certain recognition of tuberculous disease of the intestine is not easy, indeed many times an impossible task. . . The thesis may be upheld that tuberculous ulcers under our present methods can run an unrecognizable course."

In speaking of the hypertrophic form of intestinal tuberculosis, Mueller says, in the broadened intestinal loop filled with fluid contents one can see in the roentgen picture the horizontal fluid level with overlying gas. "If it is a question about a complete obstruction or a very narrow stenosis, one can see little because the meal does not move forward in the usual manner. Then the opaque enema is of help. It is to be noted that an ulcerated cecum and colon are in a condition of strong irritability and motility and that for this reason the column of the contrast meal usually shows in this place an interruption (Stierlin)."

When we consider that the whole subject of the diagnosis of intestinal tuberculosis is dismissed with this meager, wholly insufficient statement by a writer supposed to be an authority upon the subject, and ignored by such German authorities upon tuberculosis as Brauer, Schröder and Blumenfeld, it becomes apparent at once that in Germany the importance of the roentgen-ray diagnosis of intestinal tuberculosis has been completely overlooked.

Schlesinger, as late as 1922, says that roentgen-rays in diagnosis of ulcerative colitis compare in importance with occult blood in gastric or duodenal ulcer—valuable, if positive, of little aid, if negative. Such an opinion from an expert German roentgenologist is of much interest.

The same state of affairs exists in England, where, as far as we can gather, no idea of the modern diagnosis of intestinal tuberculosis has as yet suggested itself. In France, as we have noted, and also in Spain, one or two articles have mentioned the subject but failed to give evidence of its importance or to impress those who have read them. Gant, in a three-volume work on the diseases of the rectum, anus and colon, published in 1923, fails to mention the use of the roentgen-ray in the diagnosis of ulcerative colitis, showing that

even in America recognition of its value is extremely limited. Flemming-Møller in 1922 says that Stierlin's sign is of no great value, and that weakening of the shadows of the cecum and ascending colon, deviations of form and contour as in cancer of the stomach, thickening and loss of elasticity, of expansiveness, are more important. If this article represents the view of the Scandinavian profession, it too has overlooked the importance of Stierlin's work and has still much to learn about the diagnosis of intestinal tuberculosis. If an article, published from a Geneva clinic by Ferrando and Saloz, represents medical opinion in Switzerland, even in his own country Stierlin's work has passed unnoticed.

The scepticism with which our work was received convinced us that all previous work had been of little or no practical value and much work had to be done by us before a few workers in tuberculosis even attempted to confirm our methods.

CHAPTER II.

THE ANATOMY OF THE INTESTINES.

The intestines, that portion of the alimentary tract lying between the stomach and rectum, varying greatly in length (even 100 per cent), are divided into two parts, large and small, readily separated by gross anatomical features and serving different physiological functions.

THE SMALL INTESTINE.

The small intestine is composed of three parts, duodenum, jejunum and ileum. The jejuno-ileum is approximately 22 feet long, varying from 15 feet 6 inches, or even 9 feet 7 inches (3 m.) to 31 feet 10 inches, of which 9 fall to the jejunum and the remaining 13 to the ileum. The jejunum (signifying empty) is wider, about 1.6 inches (4 cm.), in diameter, heavier per unit length, thicker and deeper pink in color than the ileum and contains larger villi and more valvulæ conniventes, which are palpable through the walls. It also contains fewer bacteria in its contents (Cushing and Livingood). The coils of the jejunum lie in the upper part of the abdomen and run more or less transversely, while the ileum lies lower and the coils run more or less longitudinally.

Meckel's diverticulum, occurring in 2 per cent of subjects, is usually 2 inches long, $1\frac{1}{2}$ inches wide, and arises from the ileum about 40 inches (1 m.) from the ileo-cecal valve.

The study of the finer anatomy of the small intestine shows that the walls consist of four coats, mucous, submucous, muscular and serous. The mucous coat consists of a single layer of columnar cells on a basement membrane, with here and there, especially over the villi, mucus-producing goblet cells, and a double layer of smooth muscle fibers (outer longitudinal, inner circular). Migratory leukocytes are always found between the epithelial cells. The villi, minute projections of the mucous coat, are more numerous in the jejunum than in the ileum, and contain absorbent (lacteal) and bloodvessels, and muscle fibers which shorten or lengthen the villi. The valvulæ conniventes, consisting of folds of the mucous membrane, increase the absorption area of the mucous coat, retard

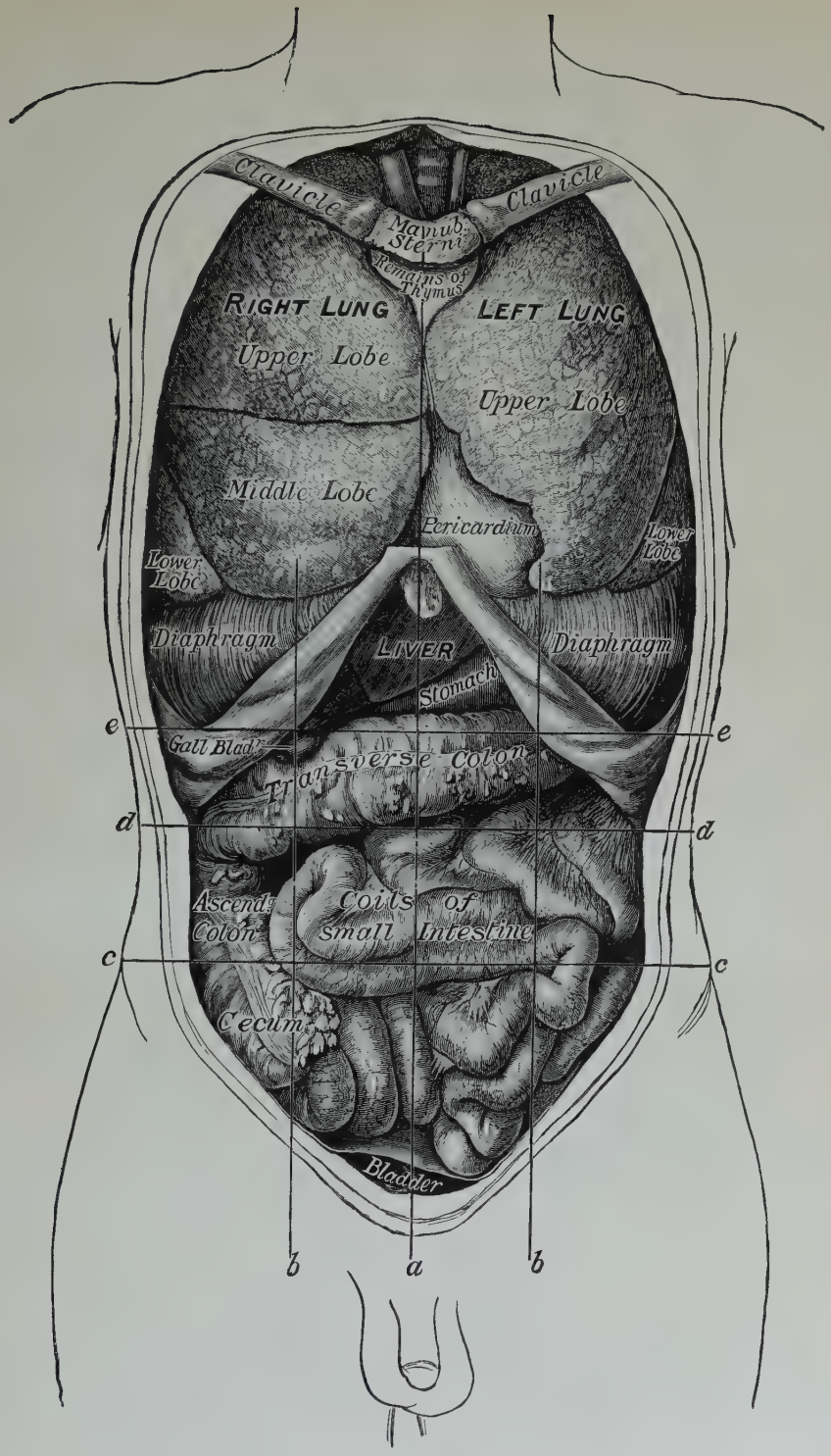

FIG. 1.—Front view of the thoracic and abdominal viscera. *a*. Median plane. *b b*. Lateral planes. *c c*. Transtubercular plane. *d d*. Subcostal plane. *e e*. Transpyloric plane. (Gray.)

the passage of food and are not obliterated when the intestine is distended. The glands of Lieberkühn are found throughout the large and small intestines and probably aid in replacing the epithelial cells, which are lost from time to time.

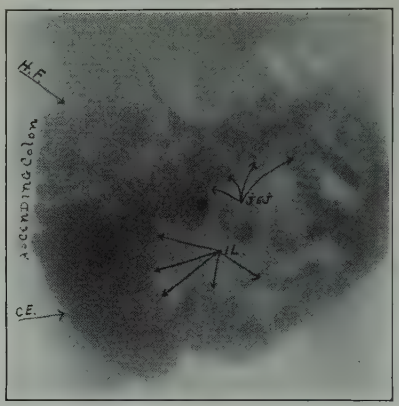

Fig. 2.—Normal small intestine. *Ce.*, cecum; *H.F.*, hepatic flexure; *Jej.*, jejunum; *Il.*, ileum.

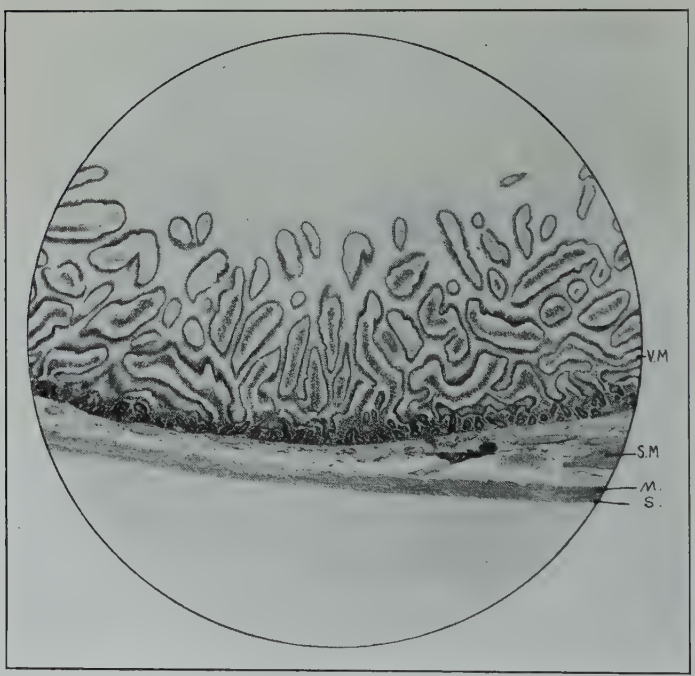

Fig. 3.—Normal small intestine. *S.*, serosa; *M.*, muscularis; *S.M.*, submucosa; *V.M.*, villi of mucosa.

The submucous coat contains the blood and lymph vessels and Meissner's plexus (plexus submucosa). While lax, this coat allows displacement of the valvulæ conniventes only in the lower part. It is composed of loose, filamentous areolar tissue and is the strongest coat of the intestine.

The muscular coat consists of an inner, thicker (2 or 3 times), circular, and of an outer and thinner, often imperfect longitudinal layer. It is better developed in the upper than in the lower end of the small bowel. Between these layers lies Auerbach's plexus (plexus myenterica).

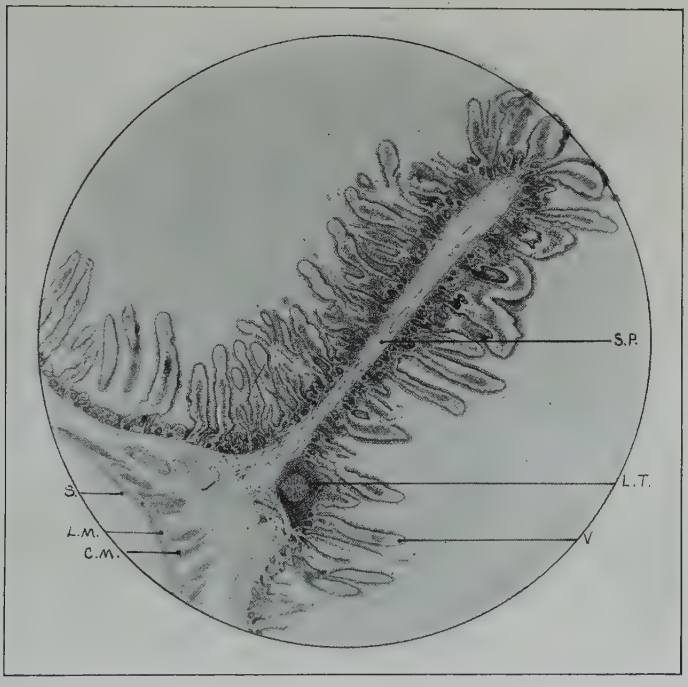

FIG. 4.—Normal small intestine—valvula conniventes. *V.*, Villus; *L.T.*, lymphoid tissue; *S.P.*, substantia propria; *S.*, serosa; *L.M.*, longitudinal muscle coat; *C.M.*, circular muscle coat.

The serous coat consists of peritoneum, a fibro-elastic stroma covered with endothelium.

Lymphatics.—The lymphatic tissue occurs as solitary follicles or nodes, or is collected in masses known as Peyer's patches, or aggregate follicles. The solitary follicles vary greatly in number and size in different individuals, in some being numerous and in

others scarce. These nodules are more or less pear-shaped, with their blunted sharper ends projecting toward the mucous membrane and their rounded ends lying in the submucosa. They are usually scanty in the upper part and more numerous in the middle and lower parts of the small intestine. Peyer's patches occur usually in the lower half of the small intestine and are most numerous and largest in the lower ileum. They lie opposite the mesenteric attachment and are usually placed lengthwise in the bowel, and while well marked in the young, become indistinct in middle life, and may disappear altogether in advanced life. They are usually about 30 in number, but as few as 18 or as many as 81 have been noted. The lymph vessels begin at the ends of the villi, collect in the submucosa, break through the wall opposite the mesenteric attachment and run around in the subserosa to the mesentery, where they are pressed to one side by the lymph glands, join and so decrease in number, and finally enter the lymph gland.

Nerves and Nerve Plexus.—The nerves of the intestine come from the solar plexus and consist of medullated and non-medullated fibers accompanying the superior mesenteric artery from the cerebrospinal and sympathetic systems. They penetrate the outer muscular layer and here, between the longitudinal and circular muscular fibers, is formed the myenteric plexus of Auerbach, which consists of both kinds of fibers as well as microscopic sympathetic ganglia. Fibers run obliquely through the circular muscles and connect in the submucosa with the plexus of Meissner. From this plexus non-medullated fibers run to the mucous coat and villi, forming periglandular and subepithelial networks. They also supply the muscle, where Berkeley, who worked with mice and dogs, thought knobs or special end-organs existed. Similar endings occurred close to or in the epithelial covering of the villi.

The connection of these two plexus with the lower seven intercostal nerves, distributed to the skin and muscles of the abdomen, explain the abdominal tenderness and rigidity, which often precede an extension of the inflammation from the visceral to the parietal peritoneum; while the reference of the early pains, no matter where the seat of the peritonitis, to the epigastrium or umbilicus (*i. e.*, to the solar and superior mesenteric plexus), the paralysis or paresis of the intestines, so common in peritonitis, causing stasis, putrefaction and fermentation of the intestinal contents, and the vomiting, at first reflex and later due to the reversed peristalsis, are closely connected apparently with changes in the two plexus.

Auerbach's or the myenteric plexus, lying between the two mus-

cular coats of the intestines, receives fibers from the vagus and sacral nerves as well as from the splanchnics (*i. e.*, from both sympathetic and autonomic systems) and contains many ganglion cells. More recently nodal tissue cells, similar to those in the heart, have been described, (Keith, L. R. Mueller). These branching cells, apparently continuous with certain groups of muscle cells as well as with the branched processes of the ganglionic cells, are intermediate in character between nervous and muscular tissue. They are said to be more in evidence near the ileo-cecal sphincter. Certain large ganglion cells have axons which reach the surface of the mucous membrane. The mesh of the plexus becomes coarser and the ganglion cells and the sympathetic neurones fewer as one passes down the duodenum to the ileum (Gerlach).

It is of interest to note that the number of afferent nerve fibers to the viscera of a cat are equal in number to those present in a single posterior spinal root. It is most likely, therefore, that the intestine is much less sensitive to painful stimuli than the skin. Most of the afferent nerve fibers of the bowel end in the muscular coat.

THE LARGE INTESTINE.

The large intestine, usually 4 feet, 6 to 8 inches (1.5 m.) long, varying from 3 feet, 3 inches (1 m.) to 10 feet, 6 inches (3.25 m.), consists of cecum, ascending colon, hepatic flexure, transverse colon, splenic flexure, descending colon, iliac colon and sigmoid flexure. The sigmoid flexure or colon is often spoken of as the pelvic colon. Like the small bowel, the diameter and capacity decrease from above downward, *i. e.*, the cecum is the broadest and largest part of the colon. The colon is said to increase 20 per cent in length from the twentieth to the eightieth year but, apart from this, variations in the length of the intestine, associated possibly with body length, age and disease, do not exceed those due to sex alone, *i. e.*, 10 per cent (Bryant). In the normal adult the capacity of the colon is 40 to 50 ounces (1200 to 1500 cc.).

The cecum is the blind end of the large bowel below the junction of the ileum and colon. It is $2\frac{1}{2}$ to 3 inches (6.25 to 7 cm.) long and about 3 inches (7.5 cm.) wide. Two main types exist; one, the persistent fetal type, occurring, some believe, in 6 to 9 per cent of cases, but in our experience much less frequently, is shaped like a cornucopia, bent to the left, with the tapering end terminating in the appendix; the second, the usual type, in 91 to 94 per cent or more of cases of adults, is due to the down-growth of that part of the cecum

between the anterior and exterior muscular bands, so that a pouch is formed, which becomes apparently the apex of the lower part of the cecum. In this type the appendix arises posteriorly near the junction with the ileum.

The cecum, more superficial than any other part of the large intestine, lies usually in the right iliac fossa, more below than above the level of the anterior superior iliac spine. It may remain in the fetal position at a point above and to the left of the umbilicus or be found in a second fetal position under the liver or indeed it may be

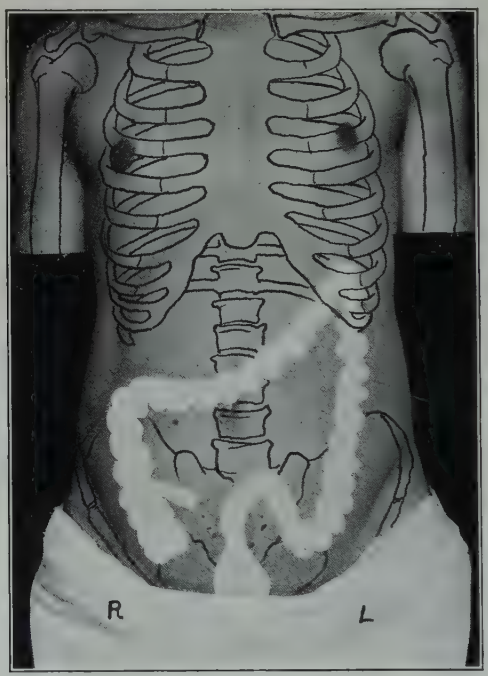

FIG. 5.—Barium filled colon.

arrested at any point in its descent from this locality. On the other hand and not rarely, it hangs over the edge and into the pelvis. At times it is freely movable or even turned up over the ascending colon. It must not be forgotten that the cecum is rudimentary in man.

The ileum empties into the colon by a round orifice, placed internally and somewhat posteriorly. A deep furrow may pass around the colon at the upper edge of the ileum and serves to separate the cecum and colon. The ileo-cecal valve and sphincter is usually

competent to prevent the return flow of fecal matter from the colon into the ileum, but gas under light pressure may do so. It is no unusual thing, however, to have a barium enema pass into the

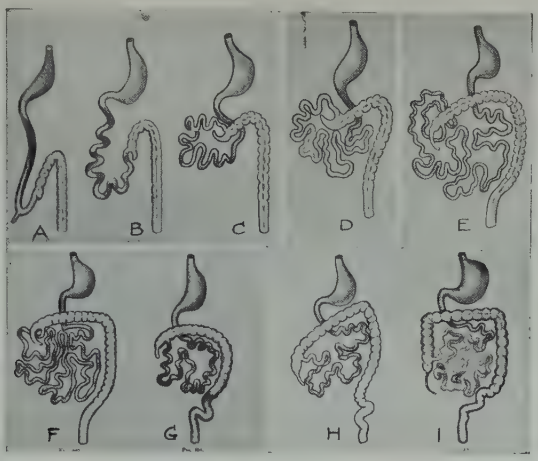

FIG. 6.—Developmental rests. Series of schematic figures illustrating stages in rotation of the intestinal canal. (Courtesy of R. A. Payne and F. C. Trahar.)

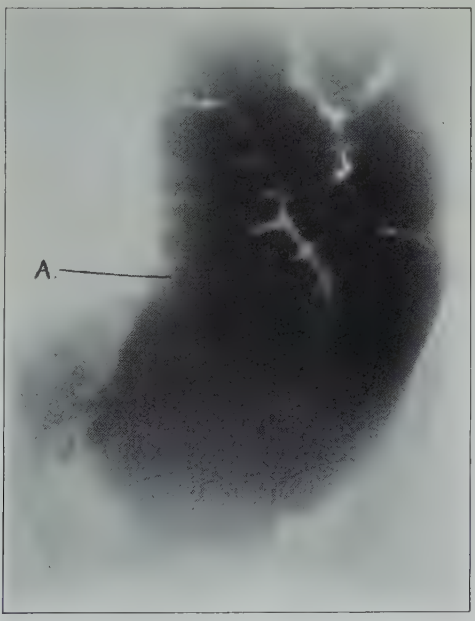

FIG. 7.—Non-rotation of the colon. (Case 7190.) Roentgen finding after enema. Ascending colon (*A*) slightly to the left of the midline. Incompetent ileo-cecal valve.

ileum, when it is allowed to flow into the rectum from a height of from 12 to 15 inches. The circular muscular fibers in the segments of the valves composing the sphincter are weak. In adults the valve lies 3 to 5 cm. internal to and above the anterior superior iliac spine. (See p. 51).

The ascending colon extends from the cecum to the under surface of the liver, where it bends to the left and backward to form the hepatic flexure. From this point the transverse colon runs to the

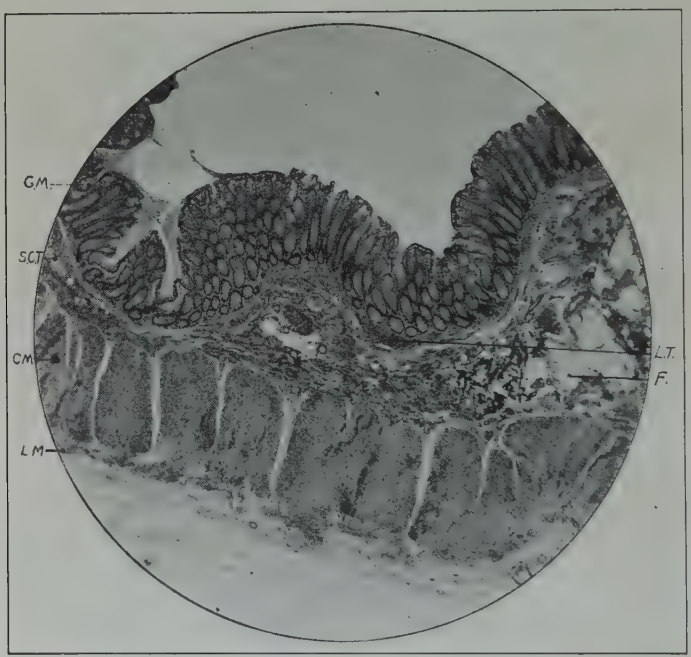

Fig. 8.—Normal colon (longituinal section). *L.M.*, longitudinal muscle coat; *C.M.*, circular muscle coat; *S.C.T.*, submucous connective tissue; *G.M.*, glands of mucosa; *F.*, fat in submucosa; *L.T.*, lymphoid tissue.

splenic flexure, which lies higher than the hepatic flexure and behind the cardiac end of the stomach. This flexure is often placed so high that the distal part of the transverse colon lies in close contact with the front of the descending colon. From this flexure, the descending colon extends to the crest of the ileum, where the ileac colon begins, which in turn changes into the pelvic colon (sigmoid flexure) at the superior aperture of the lesser pelvis and ends at the middle of the third sacral vertebra where the rectum begins. Except for the hepatic and splenic flexures, the whole colon may be palpable and

an inflamed and thickened cecum may be mistaken for a diseased appendix. An abnormal course of the colon is common. One or both flexures (hepatic and splenic) may be absent or the transverse colon may be so long, due possibly to lack of development of longitudinal muscle, that it touches even the pubis. A double fold, an accessory loop, occurs more often on the left than on the right.

The finer anatomy of the large intestine presents few changes from that of the small gut. There are no villi or valvulæ conniventes. There are no Peyer's patches and the lymphatic tissue exists only as solitary nodules, largest and most numerous in the cecum. The longitudinal muscle fibers do not form a continuous layer but are grouped into three flat bands, exterior, interior and anterior, about $\frac{1}{2}$ inches (12 mm.) wide. These bands are shorter than the other coats and help form the saculi (haustra) of the cecum and colon. The circular muscle exists in a thin layer, largely between the bands.

CHAPTER III.

THE NORMAL PHYSIOLOGY OF INTESTINAL MOVEMENT.

RATE OF MOVEMENT IN THE INTESTINES.

The movement of the partially digested food and its remnants through the bowels of man and animal has been studied in many ways and by many methods, but none has been so helpful as the observation of food impregnated with some substance impervious to roentgen-rays, usually barium as it is much cheaper than bismuth. By this means some of the stomach contents (barium meal) are seen to pass through the pylorus shortly after ingestion and fill the first part of the duodenum, called by L. G. Cole the cap. Its passage through the rest of the duodenum and the jejunum is observed less often except under pathological conditions. The jejunum has earned its name (meaning empty) for with part of the meal in the stomach and a considerable quantity in the lower ileum it is rare to visualize a well-filled jejunum, or, for that matter, even the ileum, until the meal reaches the last few feet. The barium can be noted at the ileo-cecal sphincter as early as three and a half hours and begins usually to pass into the cecum in about four and a half hours. (This relates to the period of time which has elapsed since ingestion.)

The length of time the ileum contains part of the meal depends, of course, upon when the last of the meal leaves the stomach, and ileal stasis, as Cole has often remarked, is directly dependent upon this factor. It is usually visualized from three and a half to twelve hours.

The ileo-cecal sphincter was described first by Achillini and later by Bauhin in 1579 as a valve and looked at from the side of the cecum it much resembles one and probably acts like one. Its function is apparently two-fold: (1) It acts as a sphincter to prevent the chyme from passing out of the small into the large bowel until such time as the digestion has been completed and absorption of most of the products of digestion has taken place. (2) It acts as a valve to prevent the backflow of feces into the small bowel.

This was first put forward by Good in 1822. Keith (1903), from an anatomical study, arrived at the conclusion that a true sphincter existed at this point and Elliott the next year confirmed this view experimentally. He cut the splanchnic nerves and found colonic contents passed freely into the ileum. This does not disprove the valve action, which, as in other structures is dependent also upon the tone of the muscles about the opening. A barium enema flowing into the large bowel at a pressure of 12 to 15 inches will, in some cases, pass easily into the ileum.

Rutherford (1914), who had an opportunity to observe the ileo-cecal sphincter through a large cecal fistula, noted it as a large, smooth, hemispherical papilla, about 1.8 cm. in diameter, projecting about 1 cm. into the lumen. The orifice, when the peristaltic wave from the ileum reached it, relaxed to permit 5 cc. of fluid feces and at times some gas to pass, and then closed to form a dimple in the papilla. There was no sign of a frenula.

After part of the meal has been retained in the terminal ileum about one to one and a half hours, it is forced out in squirts by peristaltic waves of considerable force into the cecum, which is filled passively, and becomes visible on an average in four and one-quarter hours (three and a half to five hours). In six to six and a half hours (five to eight hours), the head of the column has reached the hepatic flexure. In nine hours (seven to fourteen hours) the meal is at the splenic flexure; in eleven hours (eight to sixteen hours), at the end of the descending colon; in twelve hours it is seen in the pelvic colon (sigmoid flexure). During defecation, all of the contents of the large bowel lying beyond the splenic flexure are discharged and, at times, the contents lying in the distal half of the transverse colon may be included. As the head of the column reaches the splenic flexure in nine hours, remnants of food eaten nine hours before may be discharged at that time, or, if, as is usually the case, the bowels move but once a day, it may not be evacuated until twenty-four hours later or at the end of thirty-three hours. The barium meal should be completely eliminated in thirty-six hours and the colon found empty. *Normally both the ileum and transverse colon never contain barium while the cecum and ascending colon are empty.* Newton, in a study of 58 healthy individuals seven hours after a bismuth meal, found the head of the column was in the cecum in 4, in the ascending colon or hepatic flexure in 12, in the transverse colon or splenic flexure in 31, and in the descending or pelvic colon in 11.

The terminal ileum should be empty in four to six hours after the

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meal has passed out of the stomach, which usually occurs in four or five hours. It is of interest to note that the terminal ileum retains food longer than the stomach.[1]

Observation and empiricism are in medicine usually the forerunners of scientific explanation. So it has been with the study of the progression of food through the bowels. The subject may be somewhat more clearly understood, if we consider first the various types of movement occurring in different parts of the intestine, and then discuss the underlying mechanism that coördinates these movements.

TYPES OF MOVEMENT.

In the small intestines there are two main types of movement with different aims in view. On an average, the contents move through the 22 feet of the small intestines at a rate of $1\frac{1}{4}$ inches per minute and, as has been stated, reach the ileo-cecal sphincter in about three and a half or four hours. This is brought about by peristaltic movements, occurring in what have been called rapid peristaltic rushes, which, continuing for about a second, convey the food onward for a few inches. In a dog a weight of 5 to 8 gm. checked the onward movement of food and caused colicky movements and uneasiness.

Much of the digestion occurs of course in the small intestine and it is necessary that the intestinal digestive juices be thoroughly mixed with the food, and, furthermore, that the chyme be brought over and over again in contact with the various folds and crannies of the intestinal wall. This is accomplished by a second type of rhythmic movement, termed segmentation, which has been noted in the duodenum, but occurs more frequently in the lower ileum, and is produced by sharply localized constrictions of the wall, dividing a portion of the bowel into segments. A churning motion occurs in the various segments thus formed, which, after a time coalesce with others in order to reform into new segments and so on over and over again. In the cat new segmentations appear every three seconds. The diagram on p. 53 will make this clear. Segmentation is held to aid also in the circulation of the blood and lymph.

Beside these two movements another movement, also rhythmic, has been noted and termed pendular or swaying motion.

In the large bowel several types of movements also occur. The peristaltic movement here again propels the contents onward and

[1] Those interested in this problem should consult Hurst: Constipation and Allied Intestinal Disorders, London, 1919.

moves them fairly rapidly for 10 or 12 inches in a very few seconds. Such movement has been called mass peristalsis. Just before this occurs the folds of the bowel wall, called haustra, which project often well into the lumen, apparently disappear, the mass previously irregular in outline assumes now an elongated form with smooth edges and rounded ends (sausage shape) and may pass on rapidly, for instance, from the hepatic to the splenic flexure.

This movement of the contents of the large intestine, which can be readily visualized on the fluoroscope, curiously enough long escaped detection, due probably to the fact that such movement occurs only 3 or 4 times a day, chiefly after eating, and in all probability in many persons whose bowels move once a day, mainly after breakfast and just previous to the desire for defecation.

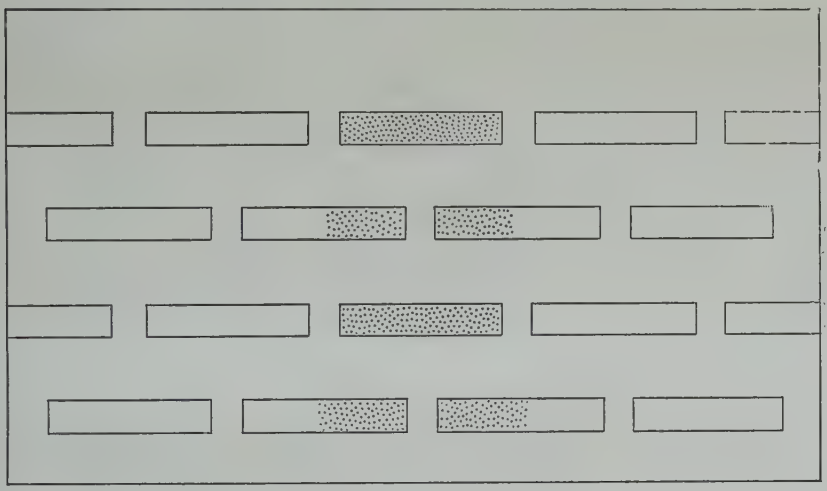

Fig. 9.

The haustra divide the colon and cecum into pouches or sacks and, here, as in the small intestine a churning motion has been described. This occurs particularly in the distal half of the colon, where the feces begin to assume the consistency they have when evacuated. Much of the water is said to be absorbed in the pelvic colon.

Slow propulsion of small thumb-sized masses has been described.

A third type of movement of the large bowel has been more recently described as reversed peristalsis or anastalsis. While few have observed peristalsis, fewer still have seen this reversed action. Case has noted it in a number of instances. It apparently began in

a small contracted ring near the junction of the first and second quarters of the transverse colon. An indentation of up to about $\frac{3}{4}$ inch was seen to run almost to the junction of the ascending colon and cecum at a rate of five a minute for five minutes. It is still a question how often it occurs normally and how important this movement is in man, in whom little or no digestion occurs in the large bowel. That reversed peristalsis can exist throughout the whole intestinal tract cannot be denied, for patients have vomited an enema within ten minutes of its injection and even portions of suppositories have met the same fate (see F. P. Weber, Strauss, Jaccoud). Case has seen it in the descending colon.

It has been noted that, if tracings are made of the large bowel, it is found to undergo constantly some change in shape, which, however, is thought to have to do only with the mixing of the contents, not with their forward movement. A pendulum movement has also been observed.

It is difficult to explain entirely satisfactorily how the orderly movements of the meal through the intestinal tract are brought about. It is most simple to assume that a contractile impulse is passed from fiber to fiber in the intestinal muscles. But this fails to explain why in a reversed loop of the intestine attempts are made by the loop to force the contents against the usual flow. Recently a "gradient idea" has been advanced by Alvarez, which assumes that each portion of the intestinal tract has a specific power and rhythm of contraction. They are most highly developed in the duodenum and the jejunum, which, as we have seen, hurry the meal rapidly throughout their whole extent, and become less marked in the ileum and large bowel. In other words, the muscle at the orad (proximal) end of a bolus contracts more forcibly than that at the distal end and consequently the food is forced through the intestine.

Formerly much importance has been attached to the so-called law of the intestine, or the myenteric reflex, which, stated briefly, is that a wave of relaxation precedes the wave of contraction, for otherwise the contraction would squeeze but might fail to move forward an object in the lumen. This is the distinguishing feature of peristaltic contraction (Starling). It is not always easy to prove that such conditions do exist and so some question this rather widely accepted statement. It is well known that the intestines or, indeed, most smooth muscle contracts at that point where the tension (distention) or pressure is greatest, which, indeed, might readily explain the law of the intestine, the peristaltic rushes, and the production of segmentation and churning.

THE NERVOUS MECHANISM OF THE INTESTINES.

Much work must still be done before the nervous mechanism of the intestinal movements can be said to be satisfactorily and finally explained. In the first place, it can be stated that intestinal peristalsis is largely independent of the extrinsic nerves and continues after all are cut. Excised intestines, short segments of the bowel, or, indeed, bits of muscle removed from the wall show peristaltic and rhythmical contractions when placed in oxygenated Locke's solution. It is of interest to note however that the rhythmical contractions occur only after the development of Auerbach's plexus and the longitudinal muscle, but that intestinal muscle contracts while not in conjunction with its nerve tissue.

It is difficult to conceive that Auerbach's plexus plays as small a part in intestinal movements as some suggest. It undoubtedly aids in conduction of stimuli and in the coördination of movement. All motor impulses may not arise in this plexus for the muscle fibers contract rhythmically when isolated from the plexus, but motor impulses in the intact intestinal wall are closely connected with the plexus. Some even hold that the whole bowel movement is purely reflex, due to sensory impulses arising most often in the mucous membrane, and completing its nerve arc to the muscle through the plexus. It is interesting to recall that some think both motor and sensory ganglion cells, as well as nodal tissue cells, occur in the plexus. The plexus is closely connected with other parts of the nervous system by various extrinsic nerves, which modify through various external influences the intestinal motility.

Keith studied Auerbach's plexus carefully in a series of animals and concluded that there was nodal tissue in the intestines. The total result convinced him that the myenteric plexus is not a simple structure composed of merely nerve cells and nerve fibers, but one of a composite texture. "There are ganglion cells in the plexus and there is an ample network of particularly fine fibers. But there is also an abundant third element in the plexus—the branching intermediate cells, which have been already mentioned and which appear to become continuous with processes of certain groups of muscle cells on the one hand and with the branched process of ganglionic cells contained in the nerve fibers of the plexus on the other. Indeed, it is often difficult to say whether a certain intermediate branched cell is to be regarded as a muscle cell or as one of the cellular elements of the plexus, so alike are they in form and in staining reaction."

Speaking of its function, he states that as a working hypothesis,

"the myenteric plexus represents a nodal and conducting system. The intermediate branched cell is capable of assuming the appearance of either a nerve element or of a muscle element. It is like the nodal tissue in the heart. We believe, as in the heart, efferent nerves, which influence contraction, tone and rhythm of the bowel, would not end directly in the musculature of the bowel but in the tissue of the myenteric plexus, and that it is through the intermediation of the intermediate cell elements of the plexus that the splanchnics and vagi effect their influence on the movements of the bowel.

"I observed that the plexus was very plentiful in the distal part of the transverse colon, in the descending and iliac colons. It is well known to be highly developed in the rectum. Nowhere did I find any special plaque or area of nodal tissue, but everywhere—particularly in the descending colon—there was an abundance of intermediate cells. I was inclined to correlate the tonic properties of the transverse and descending colons with the richness of their myenteric plexus. I noted, too, that in cases of colitis there was the same form of fibrosis of the muscular coat as one sees in the myocardium of diseased hearts—a fibrosis in which meshes of the myenteric plexus are often involved. I noted, further, that in cases of colitis and of intestinal stasis there were structural changes in the cellular elements of the plexus.

"Dr. Alvarez found that the first segment of the jejunum has a slightly higher or faster rhythm than that in the last part of the duodenum, but that from the first part of the jejunum to almost the last part of the ileum the rate of rhythm decreased. At the last part of the ileum, if a piece of the ileo-cecal junction were left attached to it, the rate actually increased, but only if the ileo-cecal junction were left attached. That fact interested me, because it was explicable—if I were right in regarding the ileo-colic collar as a nodal center—as a pacemaker for the cecum and ascending colon.

"Disturbance in any one segment upsets the rhythm in all the segments. Bayliss and Starling observed that distention of the duodenum inhibited the action of the ileum; surgeons are familiar with the fact that a duodenal disturbance upsets the rhythm of the stomach. From the facts, already mentioned, it is easy to see that disturbance in the excitability and rhythm of the pacemaker of the cecum will be reflected to the lower ileum."

Auerbach's plexus controls, according to Durig, the segmental movement of the intestine, as well as the course of the peristaltic waves, which move the intestinal contents from one section to the other. These waves in the large intestine take place over large areas

and can also move the contents backward. It controls as well the tone through tonic contraction. Often segments of the large intestine can be so narrowed that the contents are completely pressed out. While on the other side, especially in the small intestine, the contents are divided into clumps. The muscles of the whole intestinal tract show tonic innervation, especially that part of Bauhin's valve and Keith's neuromuscular ring, (questioned by some), which is placed on the limit between the first and second thirds of the transverse colon and has the task of delaying the passage of the contents, acting similarly as the first part of the duodenum.

The important stimulus for Auerbach's plexus, he notes, is the intestinal contents. The vagus and in the lower part of the colon, the pelvic nerve, exerting their influences as parasympathetic nerves and acting in a motor way, increase also the tonus through their action on the plexus. The fibers coming from the splanchnic, which pass through the solar ganglion, or the superior and inferior mesentary plexus, inhibit this working. Its irritation therefore quiets the intestine and lessens tonus. Only at Bauhin's valve is the sympathetic (where it acts as in the case of the pylorus) the important nerve governing contraction; the vagus on the contrary is the inhibiting nerve. Both nerves stimulate tonicity.

Cutting off the stimulation of the vagus and sympathetic nerves, Durig holds, curbs or lessens the autonomic movement in the intestine itself and the tonic contraction, as in the heart. If the vagus predominates as is the case in heightened vagotonus, diarrhea or painful persisting cramps can occur. In the same way acts the paralysis of the sympathetic, *i. e.*, the influence of the normal tonic stimulation of the vagus predominates and so may produce symptoms. The removal of stimuli, whether through the change of reflex stimulation, or through cutting off the autonomic system in other ways, may be the cause of the emptying disturbances. Such processes dominate by far over all those produced through muscle weakness, and lessen activity of the intestine, and perhaps are set free through arteriosclerotic intestinal changes in old age. What was once indicated as obstipation is for the most part to be attributed to a low irritability of Auerbach's plexus, which produces as a result insufficient peristaltic waves. The vagus fibers pass directly to the muscle fibers on the one side, and on the other they act indirectly through their connection with Auerbach's plexus. While the direct fibers lead chiefly by their stimulation to local spastic and atypical contractions, the other fibers produce through Auerbach's plexus an increased tonus and normal peristalsis. Thus

it happens that all poisons and all stimuli, which work on the vagus and intestinal plexus, produce heightened peristalsis, spoken of as painful spasm and colic. In this way fright, anguish or cold and many medicines set free heightened peristalsis and diarrhea. Measures which paralyze the vagus or lessen its irritability, such as warmth, diathermy, bodily rest, and quiet, may, therefore, prevent diarrhea or spasm due to the intense action of the vagus.

The glands of internal secretion may influence the intestinal motility in a general way through the action of the thyroid and of the adrenal. The hypophysis may act through its internal secretion chiefly on intestinal contraction, especially on Auerbach's plexus, and set free through its hormone well ordered, strong peristalsis. How far the changes in intestinal motility affect nourishment depends upon which part of the intestine is affected. Nicotine, even a cigarette, may cause painful colic in duodenal ulcer. Meat and albumin in large quantities have a depressing action on intestinal peristalsis. Slight quantities of food are almost indifferent.

A decrease of peristalsis is produced through the prevention of irritation of the mucous membrane or of the nervous apparatus through many known means, such as decrease of the volume of food, and warmth, but, especially, rest in bed.

Chemical action of the intestinal contents and adrenalin may stimulate the nodal tissue. Thyroid extract, and extracts of the walls of the stomach and intestine may influence the plexus, while excessive smoking may depress it. Solutions of sugar, acetic, butyric, citric, lactic and tartaric acids, formed during digestion, may affect the movement of the small bowel, but apparently only slightly that of the large.

Less is known of Meissner's plexus (plexus submucosa), where the medullated nerve fibers are rare and finer, and the meshes smaller than in Auerbach's plexus, with which it is closely connected by many branches. Durig in his interesting article ascribes to it special motor workings of the mucosa, by which this membrane is enabled to escape injury from pointed foreign bodies. The tactile irritation of the mucous membrane produced by the ingesta, may be passed by Meissner's to Auerbach's plexus with which it is intimately connected by nerve trunks. The plexus submucosa is considered by some to play an important role in connection with the pump-like action of the villi and with the process of absorption. It is said also to control the pendulum movement of the small intestine.

The extrinsic nerves of the small bowel come from: (1) The vagi (bulbar autonomic supply), whose stimulation causes a contraction

or increased tone of the muscles; (2) the splanchnics (thoracic autonomic supply); and (3) branches from the superior mesenteric ganglia, both of which, (*i. e.* 2 and 3) when stimulated cause relaxation or inhibition of tone.

The large intestine receives its motor nerve supply largely through the pelvic nerves (sacral autonomic) and its inhibitory fibers from the splanchnic branches, connecting with the inferior mesenteric ganglion (thoracic autonomic).

A number of reflexes are connected with the movement of the contents of the stomach and intestines. For instance a gastro-colic or duodeno-colic reflex has been described which has to do with the movement of feces in the small and large bowels. Passage of the food out of the stomach or even odors of food can stimulate the reflex. The peristalsis is most active in the terminal ileum during and just after a meal and Hurst thinks that a gastro-duodeno-ileal reflex regulates this movement. In some irritative conditions of the ileo-cecal region there is apparently at times some gastric delay, but White was unable to produce it in cats except with strong stimulation and doubts its occurrence in man. Dilatation of the ileum causes retention of the food in the stomach (Cole).

Finally it might be mentioned that dyspnea may increase peristalsis, that shutting off the blood supply causes violent intestinal movements, that CO_2, CH_4, H_2S may also increase the movements either by distention or by chemical stimulation. Distention of the intestine however by food or gas is probably the chief cause of the motor functions, either through its action on the nerve endings or more directly on the muscle itself. There is no evidence that hormones circulating in the blood evoke motor reactions.

CHAPTER IV.

PATHOLOGICAL ANATOMY OF INTESTINAL TUBERCULOSIS.

The difficulty of producing ulcerative intestinal lesions in small experimental animals, especially apart from a generalized tuberculosis, has greatly retarded the pathological study of intestinal tuberculosis. The following discussion is based, consequently, almost entirely upon a study of the lesions in man.

Tuberculous lesions in the intestine occur most frequently in the ileo-cecal region. As the infections are brought about in nearly all instances from direct contact with the contents of the bowel, it is to be expected that the first lesions would appear in the submucosa of those parts, where the contents remain longest in contact with the walls, and where the lymphoid tissue is most abundant, *i. e.*, the terminal ileum and the cecum. Such is usually the case, the ileo-cecal region being involved in about 85 per cent of all cases and, while any part may be affected, the frequency of involvement decreases as the tract is ascended to the stomach or descended to the rectum (see p. 107). Again, the earliest lesions are found usually in this region, which emphasizes its importance in diagnosis.

THE PATHOLOGICAL HISTOLOGY OF INTESTINAL TUBERCULOSIS.

As tuberculosis begins practically always in lymphoid tissue, we should expect to find the earliest and usually the most pronounced changes in the lymphoid follicles in the intestines, either in Peyer's patches or in solitary follicles.

The earliest lesions, whether in the form of miliary or larger tubercles, which have progressed from the central portion of a follicle, or of minute ulcers, are situated on the summits of the lymph follicles. In only 1 case were they associated with bloodvessels (Schwatt).

Such lesions, in the form of gray translucent nodules, occur most commonly in the solitary or aggregate follicles, but can arise occa-

sionally on the border of the follicle or elsewhere. Caseation sets in before long and the nodule becomes yellowish. The contents soften and become more or less liquid to form thick puriform matter. The mucous membrane over such nodules, varying in size from a pea to a bean, which project into the lumen of the gut, is often injected, and reddened or purplish in color. At this stage the tuberculous matter may inspissate and, as some state, even calcify or, what is more usual, the mucous membrane over the apex of the swelling may necrose, turn yellow and burst to form after the contents are discharged, a crater-like ulcer. The edges of such an ulcer are usually infiltrated, flaccid and undermined.

Gottsacker, who made a careful and detailed microscopical study of the early lesions of intestinal tuberculosis, stated that it is difficult to say whether the first lesions occur in or on the periphery of the follicle. The follicle is first infiltrated with round cells, but scarcely at all enlarged. Indeed, it may be strikingly small. A larger or smaller zone about the follicle is also infiltrated and it may be difficult to determine the borders of the follicle. Within the follicle the cells lose their sharp borders, run together and form a spotty, translucent mass. In other words, they undergo coagulation necrosis, caseation and softening, which affect the whole follicle, and, of course, at times extend even further. The surrounding infiltration increases and the overlying mucous membrane is also infiltrated. It ultimately necroses, bursts, the contents empty and a small crater-like ulcer forms with swollen edges as already mentioned.

The perifollicular infiltration can be separated into 3 zones; the first composed of lymphoid cells; the second of proliferated connective tissue cells with bloodvessels; lymphoid cells with some spindle cells form the third zone, which passes over into and infiltrates the submucosa. Many polymorphonuclear cells and some giant cells are found in the periphery of the caseated area. In the granulation tissue about the follicles and, at times elsewhere, are also found giant cells. Further away from the ulceration young, fresh and, at times, incompletely formed tubercles are seen, which apparently are not found in the affected follicle. Tubercle bacilli are present most often in ulcers of the mucosa, and are less numerous in the subserous tubercles.

The small arteries have a cellular infiltration about them, their wall thickens, their lumen narrows or disappears, and the surrounding zone undergoes hyaline degeneration. The arteritis

obliterans aids in the extension of the ulceration. The veins are not affected.

In a study of 7 cases of intestinal tuberculosis with diarrhea and of 16 without diarrhea and undiagnosed clinically (all 23 having extensive pulmonary tuberculosis), Leupold found in all the cases similar changes in the mucosa and submucosa. The ulcers were chiefly in the lower ileum and cecum, but in many were present throughout the bowel. In some cases the ulcers were very few. He calls attention to the discrepancy between the anatomical findings and the clinical symptoms which occur in so many cases.

In the cases with diarrhea the ulcers were more widely extended, even beginning in the jejunum and extending well into the large bowel. Such pictures reveal of course a late stage in extensive intestinal tuberculosis. The ulcers were most frequent in the cecum and ascending colon. Only in 1 were ulcers found in the rectum. On the other hand, the cases without diarrhea had more scattered ulceration, but this by no means always occurred. In 1 case, free from diarrhea for five weeks while under observation, extensive ulceration was present. Ulcers were present in the small bowel and some in the cecum and ascending colon, many of which penetrated nearly to Auerbach's plexus. It would appear that in many cases some other factor than the mere presence of ulceration must account for the diarrhea.

The myenteric plexus was then studied, and he found the changes were more extensive and more severe in the patients with diarrhea. In these patients degenerative changes occur more often in the ganglia cells. Askanazy's inflammatory edema of the nerve sheaths was found in all 7 patients with diarrhea, as well as perineural and periganglion round-cell infiltration. In 3 of the 16 patients without diarrhea this round-cell infiltration was present, but only in the zone about the ulcers and only in a few ulcers, while in the other 7 cases it was widely spread, and in a great number of ulcers was found along some of the nerves in the septa of the circular muscle. It is difficult to say positively that such changes in Auerbach's plexus are the sole causes of the increased peristalsis and symptoms, for the mucous membrane is often widely affected by catarrhal conditions, and nerve terminals, thus situated, must also be more irritable. Sasaki also found real degeneration of the ganglion cells in Auerbach's plexus in the immediate vicinity of the tuberculous ulcers.[1]

[1] Rokitansky's excellent description should be noted (see p. 28).

GROSS PATHOLOGY OF INTESTINAL TUBERCULOSIS.

The earliest lesions, as already noted, occur in the ileo-cecal region. The terminal ileum or cecum may show only more or less numerous, scattered, projecting nodules, a few of which show on careful inspection beginning ulceration. In others the nodules are not noted but fine ulcers, seen only after the bowel is washed, may occur in varying numbers. These are usually situated over solitary follicles or occur in the Peyer's patches. In some cases the whole process can be noted; enlarged follicles covered with normal mucous membrane; others with reddened or yellowish necrotic mucous membrane; and, finally, others exuding grumous matter where ulceration has already taken place. In such instances the ulcers are small, round, with overhanging edges and rather superficial. A little later such ulcers by extension coalesce and form large irregular ulcers.

When this process occurs in a Peyer's patch, different follicles may be attacked, either simultaneously or successively. The whole patch, though possibly swollen, is, unlike in the typhoid ulcer, never affected at first. Consequently it is not unusual to see islands of mucous membrane surrounded by ulceration.

Pathological changes, similar to those found in the follicles, may extend more or less widely in the submucosa and lead to rapid extension of the ulceration, without the formation of distinct granulation tissue. In some instances, the dense, whitish, raised, irregular plaques, 1 or 2 inches in circumference, under the mucous membrane, are due to such changes.

The larger ulcers, often ultimately formed, as already noted, by coalescence of smaller ulcers, have often thickened, infiltrated, everted, indented, gnawed edges, frequently undermined, with the muscularis or more usually the submucosa forming the caseous or reddish base. This is usually more or less covered with a grayish or caseous swollen, gelatinous layer, from which spring tuberculous granulations often in a row around the edge, at times assuming the proportions of the tuberculomata to be found on the walls of the larynx. The whole base may be thickened (covered) in this way, which in reality may be considered a conservative process, rendering perforation less likely. Perforations, on account of early forming, localized peritonitis, rarely rupture into the free peritoneal space. Nodules, prone to caseation (tuberculous granulation tissue), occur often in great numbers about the ulcer and tuberculomata may spring up near it.

In the small bowel the ulceration frequently extends through the muscularis, and the serosa is frequently injected and, at times, covered with a few strands of fibrin. Under the peritoneum, over the base of the ulcer, and along the course of the lymphatics, tubercles are often seen and nodules felt. Extension of the tuberculous process usually follows the lymphatics and circular ulcers result, which form, however, is not confined to tuberculosis. In fact, tuberculous granulation tissue, limited for a long time to the submucosa, may assume this girdling form. In the ileum, however, the ulcers may follow the outlines of the Peyer patches and may assume, consequently, an ovoid form with a more or less longitudinal direction. In advanced cases the ulcers are often longitudinal.

The lymphatics are, as already noted, usually and early involved. Nodules may be formed in the lumen of some of the larger vessels, which may occlude them and bring about stagnation of chyle, which renders that lymphatic very prominent. At times, several tubercles may be seen along the course of or within a single isolated vessel, causing it also to be prominent.

The ulcerative process in the large bowel is often arrested by the muscularis and the serosa is rarely affected. Serpiginous and phagedenic ulcers have been described in the cecum and ascending colon, and in a few cases nearly the whole of the bowel from the lower end of the ileum to the rectum has been ulcerated.

Girdle ulcers are found more often in the ascending and transverse colon, but usually the ulcers are irregular and may have undermined edges. The ragged, irregular, serpiginous or phagedenic ulcers found in the cecum are due in part, Maylard holds, to secondary infection.

In some advanced cases tubercles are found in the muscular coats, not involved in the ulcerative process. The muscularis may show some fatty infiltration. About the ulcers the intestinal wall may be edematous and thickened but more often is thinned, though atrophy of the muscular coats rarely occurs.

In the large bowel sloughing and false diphtheritic membranes may occur. Some have described the simultaneous occurrence of non-tuberculous ulcerations (amyloid), but further confirmatory evidence of these is needed. Tuberculomata in the form of polyps have been mentioned.

The mesenteric glands, which almost always contain tubercles, bear no relation to the severity of the tuberculous process in the bowel, though they have been called the mirror of the intestinal

lesions (Parrot). In isolated ulcers of the large bowel they are but little or not at all affected. In his 100 cases of intestinal tuberculosis Engelsmann found these glands affected 56 times. In 34 they contained pus and were caseous in the center. In 11 cases they were greatly swollen and in 4 calcification was present. In the majority of Schwatt's cases they were tuberculous and in 10 per cent caseous.

Girode in a careful study of 29 cases, found the diameter of the bowel was variable. In 1 the small bowel was greatly dilated and the large in 2. In the majority the bowels appeared pressed down and in 2 had a reduced diameter. He measured the length in 13 cases and found the small gut averaged 6.9 meters (normal length 8 meters). One with tuberculous peritonitis was only 4 meters long. Congestive or inflammatory redness was found in 13 and was usually not generalized, but occurred in zones, more in the small intestine and about the Peyer's patches. In 8 cases the Peyer's patches were swollen as in typhoid fever. The mucous membrane is often discolored and black patches the size of a 5-franc piece may occur. The intestines may be perfectly empty. Both large and small were affected in 17, the small only in 12. He quotes Louis[1] as thinking the bowel is always affected in patients dying from pulmonary tuberculosis, and as finding in his 120 cases, tubercles in 24, ulcers in 96. Girode found infiltration in 2 cases (one 6 to 8 cm. square) but usually granulation. The annular type of ulcer, rarely complete, he found in 7 of 24 cases, the longitudinal (in a Peyer's patch) in 9. The two types can coexist. Large ulcers arise from necrosis of the mucous membrane or by coalescence of smaller ulcers. A third type of ulcer, frequent in the small bowel and first portion of the large, is irregular, serpiginous. The base of the ulcers is usually the muscularis. In the large bowel, on the other hand, the ulcers are larger, irregular, not deep; and nothing may exist exteriorly to show that advanced ulceration is present within. Large, deep ulcers with cut-off edges, deeply red, are mentioned. Lymphangitis was found in 9 cases. Important involvement of Auerbach's plexus may occur (see p. 62).

Gant, in a study of 100 cases, found 80 with superficial ulcerative entero-colitic lesions, 8 with deep ulcerative entero-peritoneal lesions, 4 with hypertrophic tuberculosis, 2 with the fibro-sclerotic "gas-pipe" of Mummery, 2 with glandular, 3 with "peritoneal," and one with generalized miliary tuberculosis.

A case has been described by Sokolowski, where a tuberculous ulcer was found in the seat of an old healed typhoid ulcer.

[1] Louis's splendid and detailed study should be consulted (see p. 22).

The occurrence of tuberculous ulcers with those of typhoid have been described by Eisenhardt, Frey, Mosler and with those of syphilis by Chiari.

A careful study of the stomach was made by Rousseff, who found in 8 cases with pulmonary tuberculosis and no intestinal tuberculosis that 7 had a normal stomach and one an unimportant change, but, on the other hand, in 26 cases with pulmonary tuberculosis and intestinal tuberculosis, he found all had definite changes (acute parenchymatous or chronic interstitial gastritis, atrophy of the glands, amyloid degeneration).

Rosenheim draws attention to the fact that other changes can occur also in the intestines, *e. g.*, common enteritis, hyperemia and swelling of the mucous membrane, enlargement of the lymph nodes, ulcers of the follicles, as well as irregularly formed catarrhal ulcers, and those due to amyloid degeneration. General amyloidosis was present in the majority of Schwatt's cases, and a similar condition in the intestine in 8. Müller holds that amyloid ulcers can occur along with tuberculous ulcers.

Tuberculosis has been thought to occur more frequently in persons with a shorter intestine, but Bryant (1924) holds that the opposite is true for both large and small bowel, basing it on a study of 38 cases (see Girode, p. 65).

An interesting case of atrophy of the large bowel in the course of a primary tuberculosis of the small bowel has been reported by Vedel and Baumel, who state also that they have had a second similar case. The patient, female, aged forty-seven years, began to be troubled with gastro-intestinal symptoms six years previously and lost much weight. Later she had colic and marked constipation but no melena. The Wassermann reaction was negative. The Meyer reaction was also negative. The roentgen-ray examination showed marked retention in the cecum and ascending colon at end of twenty-four hours and even after eight days all the barium had not been eliminated. The shadow of the transverse colon was much smaller than usual. A diagnosis of spasm was made. At autopsy a definite tuberculosis of the small bowel was found. The large bowel was free from tuberculosis but showed a submucoid sclerosis, consisting of connective-tissue fibers without cells, extending almost to the muscle which was very markedly atrophied but without sclerous infiltration. There was no evidence of previous ulceration in the large bowel.

CHAPTER V.

PATHOLOGICAL ANATOMY OF INTESTINAL TUBERCULOSIS (*Continued*). HEALING.

The intestinal ulcerations have a marked tendency to connective-tissue formation and cicatrization, but perfect healing has seldom been described up to this time, as death of the patient usually interrupts it. Attempts at healing have been described by Spillmann, Girode, Amenomiya, Habicht, L. U. Gardner, Schwatt and others. Wilson Fox reported a case with complete healing of the ulcers in the small, but not of those in the large bowel. Eisenhardt found, in a study of 566 cases, 10 complete and 55 incomplete attempts at healing of single ulcers. Healing can occur in the presence of ulcerative pulmonary tuberculosis. Kuthy and Wolff-Eisner report the case of a woman, aged seventy-four years, dying of arteriosclerosis, who had a tuberculous cavity in the lung and in the lower ileum a scar of a healed tuberculous intestinal ulcer. Habicht reports a case of healing with extensive granulations and regeneration of the mucous membrane. Brauer and Spengler report a case treated with artificial pneumothorax with all the symptoms of intestinal tuberculosis, which recovered and remained well for seven years. Such clinical observations are not uncommon.

Schwatt noted healing, well healed and almost healed ulcers in 14 cases, most frequently in the ileum and cecum (probably the site of the oldest lesions). He thinks that ulcers may heal completely and the process recur elsewhere at the same time or subsequently. Healing and progressing ulcers may occur in the same region.

The best work up to this time on the healing of tuberculous ulcers is that of Habicht, Amenomiya and Gardner. That healing does occur has been clearly shown, but the details have not been so definitely determined. Does healing take place in intestinal tuberculosis as in typhoid fever in two ways, by cicatrization and by more or less perfect regeneration of the mucous membrane?

The necrosed parts softened by inflammatory processes, Habicht believes, slough off and the ulcer cleans itself. Granulation tissue forms over the base and raises it, making it more nearly level with the edges. This tissue shrinks and, at times, not only retracts

sufficiently to decrease the size of the ulcer, but the process may even be so pronounced, that the edges are drawn so closely together that only a narrow fistulous passage remains where the ulcer was. Scarring, however, in these ulcers is usually incomplete, for, while it may occur on one side, the ulceration often advances on the other, though in a few cases such cicatrization may be complete. In some cases regeneration of the lost tissue may occur. The more superficial the ulceration the more complete and perfect the regeneration. It is highly probable that the mucous membrane and even the submucosa may be completely restored. Loss of cells or even of groups of cells must take place constantly in life and there is no reason why such repairs may not occur in slight disease. Such processes do occur in the skin.

If on the other hand loss of muscle has taken place or if extensive granulation tissue has formed, other means of regeneration can occur. In these cases the first step is the retraction of the base of the ulcer. This increases the area covered by the overhanging edges, which now rest on the base of the ulcer. Underneath these edges healing often runs a quiet course. If the cross-section of such an ulcer is studied, the base is found to consist of a more or less irregular, richly cellular granulation-tissue layer, about 2 mm. thick, lying at times upon the muscularis. The superficial layer resembles a skin-like covering, which in places may be slightly separated from the granulation tissue to which it belongs, as it is not continuous with the overhanging mucous membrane. On the overhanging edges are found Lieberkühn's crypts cut across diagonally. It has been usually accepted that the regeneration of the loss of cells in the normal intestine is made up by proliferation of the cells lining these crypts, either from all the cells of the crypt or from those in its middle portion. Similarly, cells, needed to repair, to cover the ulcerated surface, come apparently from these crypts. The cells of the crypts are pushed forward and inward and they may in places appear to be several layers in thickness; generally they form a low cylindrical epithelium, much resembling that of the intestine. At times on the base are found tags of such cells which must come from the same source, *i. e.*, the overhanging epithelium. Such rows of cells appear to stick on the base, with their broad end toward the lumen of the bowel, their sharper end buried in the new connective tissue. These cells are apparently actively dividing as they contain mitotic figures. In this covering, at times, are found what must be considered newly formed Lieberkühn's crypts, and the cells about them suggest that this was their origin. In other places shreds or tatters

of the mucous membrane, apparently cut off from all connection with epithelium, are found on the base of the ulcer. From these, of course, new cells are also formed.

So it appears that not only a new epithelial layer but also a new but irregular formation of glands can take place, and thus an actual regeneration of the mucous membrane can occur. There was also some suggestion of new lymph spaces.

Habicht further points out that it is impossible to separate what might be new follicles forming in the regenerating submucosa from young tubercles, but believes they are the former. He was not certain but that in the deeper layers some tubercle bacilli, and and even tubercles might exist.

In 7 out of 25 tuberculous ulcers, suggesting from their macroscopic appearance that healing had begun, Amenomiya found definite regenerative processes. These ulcers were definitely tuberculous as he found tubercles, giant cells, tubercle bacilli, destruction of the muscularis mucosa, and proliferation of granulations. He thought it also probable that the base of the ulcer began first to become clean. In the meantime, connective-tissue formation occurred and the base might contract more or less. In some instances the lips of the ulcer might even be everted.

From the edges of the ulcer, epithelial and glandular proliferations project or are pushed more or less widely across the base. The epithelium may pass like a bridge from the overhanging edge to the base of the ulcer, showing an implantative character. Lieberkühn's glands accompany in a similar way the epithelial layer, which has tensile properties similar to that of normal epithelium. The epithelium shows mitotic division and may be at first flat, then flat-cuboidal and finally cylindrical or may consist of many layers, resembling a true epidermis. The newly-formed glands of Lieberkühn can be bilobed, saccular and cut off short and, thus, can become cystic. So normal glands, reticulated spaces and exuberant "adenomatous" growths may result. Herzog and others have described carcinomatous degeneration in a healing tuberculous ulcer.

Girode, who described the mucous membrane very minutely, is of the opinion that it can be irritated and that the crypts in non-ulcerated areas may become bilobed and almost or fully adenomatous. During healing the muscularis thickens, hypertrophies and sends thin fibers into the submucosa. The submucosa is also thickened in scarred-over ulcers, the mucosa very thin.

In a recent article Schünemann advances a new conception of healing of tuberculous intestinal ulcers, which he claims Habicht

has entirely overlooked and Amenomiya has not really touched upon. He early calls attention to the extraordinarily great capacity of the intestinal mucous membrane for regeneration after injury, but states that complete regeneration in tuberculous ulceration is rare and that he has not found a single case in literature. He does not attempt to discuss healing by scar formation.

Schünemann has studied 14 ulcers of different sizes and of different healing tendencies from the small and large bowel. The ulcerations were studied in complete serial sections. Apart from some special staining methods for the study of mitoses, he used hematoxylin and eosin. The single ulcers showed different stages of regeneration, also a variable and strong tendency toward healing. The same sort of regeneration was found in the ileum as in the colon. He has well described and photographed some sections which he has studied. The tendency to cover the granulating surfaces is always very pronounced in the intestinal epithelium. This shows itself, indeed, in the extraordinarily strong growth of glandular loops on the borders of such defects, so that the impression is created, as other authors have also mentioned, that a polypoid growth of tissue is present. This is indicated not alone in an increase of the intestinal loops but also in their prolongation and branching. Indeed, one is inclined to speak of an infiltrating growth. As soon as the muscularis mucosa is destroyed by the tuberculous process (the muscularis mucosa tends to limit the process), a marked increase of the glandular cells from the regeneration centers of the crypts begins to occur. Loop-forming, epithelial growths penetrate into the depths and, indeed, in the direction where the most favorable conditions for nourishment exist. The epithelium of the glandular loops are for the most part many-layered. They are branched, so that one in the microscopic picture runs across astounding forms. Here are seen the numerous branches and layers of the loops far under the muscularis mucosa. The main diameter of the loop can run parallel to the long axis of the intestine. One can see a layer, lying in a defect of the granulation tissue, whose base is directed almost parallel to the lumen (long axis) of the intestine. Its upper border, which is directed toward the intestinal wall, is spread out cup-like over a split surface. This is a peculiarity of this epithelium, which is preserved always and is recognized only in serial sections, which really present before the eyes the third dimension. If the upper border of a crypt is not limited through a neighboring loop, the epithelium lies in a cup-like form over the surrounding tissue, where it spreads itself out on all sides over the

borders and has a tendency to sink into all furrows and indentations, and to grow from there again in a crypt-like manner into the depths and, thus, to form new glandular loops.

His second plate shows glandular loops of another ulceration which have penetrated from the border of the ulcer into granulation tissue, especially rich in bloodvessels, deep under the muscularis mucosa.

In the third plate the loops have penetrated on the border of a tuberculous ulcer into the depths and have already almost reached the tissue under a tuberculous nodule.

These pictures prompted him to conclude that, as soon as the destruction of the tissue through a tuberculous process begins to take place, the growth of the glandular epithelium has indeed already begun. The loops can penetrate so far into the depths of the tissue, that their deepest portions, under favorable conditions, remain uninjured by the destruction going on in the wall. The superficial layers become necrotic and are bathed with feces, but then a simple ulcerated wall is no longer present. In the remaining tissue, be it granulation tissue, submucosa or, indeed, muscle, are found remnants of the glandular epithelium, which are able to begin again the regenerative growth. The preparations mentioned suggest to the author that the loops can also penetrate into the muscles, for he has found places almost under the middle of the base of ulcers in which remnants of glandular loops were to be seen between the penetrated muscular fibers of the wall muscle. So the process of regeneration depends not alone upon the discovery, as has been held up to this time, that, after the ulcer is formed, the epithelium of the crypts is pushed from the sides over the surface of the ulcer.

He believes that one must so explain these pictures, that, simultaneously with the formation of the ulcers, a growth of the glandular epithelium begins, which can penetrate so deeply that they are never reached by the destructive process. On the cessation of the destructive process, these remnants form islands of mucous membrane, which can contribute to the covering of the whole surface of the ulcer. They are indeed so much the more suited for regeneration, as they are formed for the most part from the deepest parts of the crypts in which, according to Schafer and Cohen, proliferation centers occur.

He thus opposes the views of both Habicht and Amenomiya but acknowledges that there are some objections to this views that he cannot meet. He has not found cystic formation due to increased secretion of mucus, which occurs in the formation of dysenteric

ulcers, but did observe single small cysts on the borders of ulcers, which were brought about through the obstruction of the proliferated glandular loops.

The most recent work on intestinal tuberculosis is that of L. U. Gardner. It fails to substantiate much of Schünemann's work and suggests that the latter failed to cut many of his sections perpendicularly to the serosa. He also saw no attempts at bridging described by Amenomiya. Gardner's work is so important that it is given in detail in the next chapter. His studies deal with many cases described elsewhere in this volume and is especially interesting in view of the fact that many modern pathologists hold that complete healing never occurs in intestinal tuberculosis.

CHAPTER VI.

PATHOLOGICAL ANATOMY OF INTESTINAL TUBERCULOSIS (*Continued*). GARDNER'S WORK.[1]

This study has been undertaken in an attempt to determine the influence of treatment by artificial heliotherapy upon intestinal tuberculosis. The present communication is a preliminary report and its conclusions are in no sense to be considered as final. Before completing the study, it is hoped that a much larger series of treated cases can be studied together with material from untreated intestinal tuberculosis and from other forms of ulceration in the alimentary tract. In compiling the following data, it has always been attempted to form an unbiased judgment by examining and making written reports of the gross and microscopic lesions before collecting the data as to treatment. It is of interest to note that after those cases, showing evidences of healing, had been tabulated according to the degree of healing, few changes in sequence were necessary to make this tabulation correspond with one arranged according to the duration of treatment by the quartz mercury-vapor lamp.

The following data have been obtained from a careful gross and microscopic study of the gastro-intestinal tracts of 32 individuals dying from tuberculosis and coming to autopsy in Saranac Lake. Thirty of these cases had pulmonary cavities and 29 showed more or less intestinal tuberculosis, an incidence rate of 90.6 per cent. In 2 of the cases without cavity only microscopic evidence of tuberculosis was found in the intestine.

The technic regularly employed in this study has been to remove the intestines and open them along the line of mesenteric attachment. They are then carefully but thoroughly washed in running water and spread on a large tray in a strong light. The mucous surface is then very carefully examined with the unaided eye and with a hand lens. Blocks of tissue from any abnormal area are cut and stretched on paper for fixation in Zenker's fluid.

At least 10 blocks are sectioned and examined microscopically

[1] Dr. L. U. Gardner, Pathologist to the Trudeau Foundation, has embodied his studies in this chapter and the authors wish to express to him their grateful appreciation for many helpful suggestions.

from each case, and, in some instances, 25 or 30 have thus been studied. All cases, in which evidence of healing has been detected, have been further studied by serial section, which has sometimes necessitated the preparation of some 20,000 sections, 10 μ in thickness, from a single lesion.

MODE OF INFECTION.

It has been maintained that infection of the intestinal tract occurs through hematogenous metastasis. Where isolated groups of lesions, all of the same approximate size, occur high up in the small intestine, it is possible that such a distribution may have occurred. Two of these cases did present such a localization of ulcers, but there was no microscopic evidence of involvement of a bloodvessel. The fact, that in almost all of the cases with pulmonary cavity extensive intestinal tuberculosis was found and in addition the fact that the cases without pulmonary cavity showed only microscopic evidence of intestinal tuberculosis, would indicate that probably infection occurs from swallowed sputum. Furthermore the localization of the tuberculous lesions about the ileo-cecal valve, where the lymphoid tissue is most abundant and where the rate of movement of the intestinal contents is slowest, also favors the enterogenous origin.

HISTOGENESIS OF INTESTINAL TUBERCULOSIS.

The changes tabulated are based upon a study of 15 cases in which no treatment specifically directed at the intestine had been employed. A study of serial sections corroborates the generally accepted view that tubercle bacilli pass through the mucous membrane without producing detectable injury, and that they are then localized in the submucous lymphoid tissue, where they multiply and excite the first formation of tubercle. Accompanying the purely proliferative reaction to the tubercle bacillus is a low grade inflammation consisting of some transudation of serum and a few red blood cells, but this is slight in the cases thus far studied. The lymph-nodule tubercle caseates and extends toward the mucosal surface, which it finally penetrates. Immediately the character of the reaction changes, polymorphonuclear leukocytes and fibrin form rapidly over the surface of the tubercle, the caseous center softens and is evacuated and an intense, diffuse inflammation involves the whole area and its vicinity. Necrosis takes place in the entire

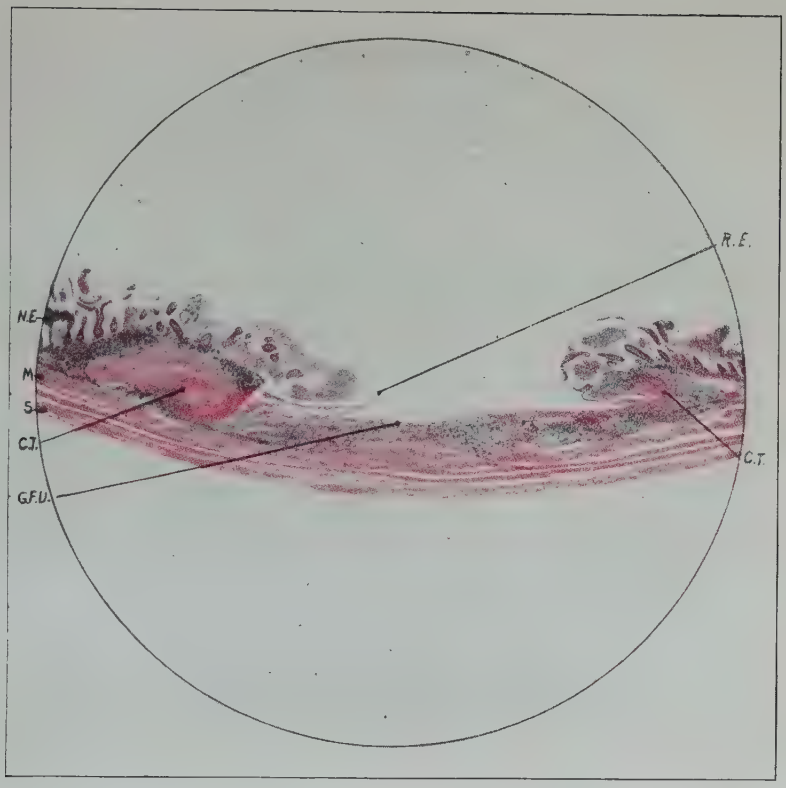

FIG. 10.—Tuberculous ulceration small intestine.

mucous membrane for a considerable distance on each side of the erupted caseous focus, and a more or less superficial ulcer is formed, which undermines the mucous membrane at its borders. Further, necrosis and sloughing very frequently destroy and remove the original tubercle, but bacilli, carried by the lymphatics, form small, proliferative tubercles within these vessels, both in the deeper tissues below and at points lateral to the original focus. These secondary tubercles then develop and, in turn, reach the surface to be evacuated and removed in a slough. In this manner the ulcer deepens and widens, and long fistulous tracts are formed in the submucosa, which reach the surface at more or less remote points, thereby forming small secondary ulcers. Lateral extension allows

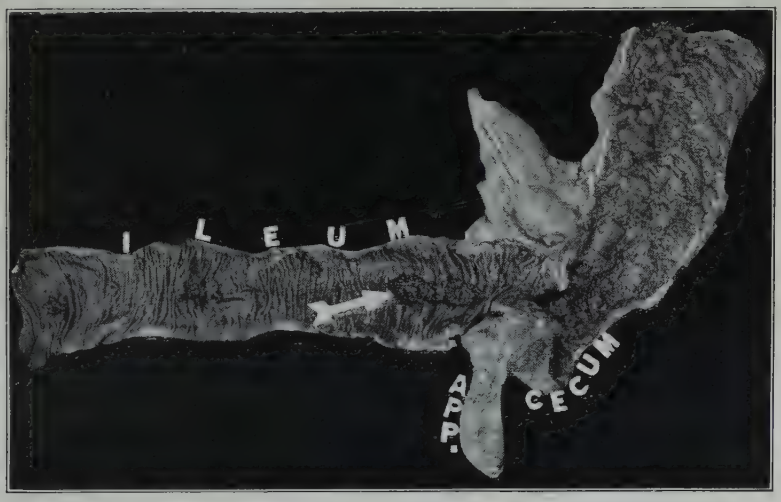

FIG. 11.—Tuberculous ulcers in the ileum and cecum (Case 151).

coalescence with the original ulcers, so that in some cases peninsulas of uninvolved mucous membrane remain connected with the underlying tissues by a slender pedicle. More remote lymphatic metastases result in the involvement of the serosal surface with tubercles, which usually are surrounded by granulation tissue. Not infrequently the lymphatic vessels are macroscopically outlined for some distance into the mesentery by distention of their lumina with tubercles. Small abortive non-caseous tubercles are formed in the mesenteric lymph nodes but they never attain macroscopic proportions. No specific changes have been observed in the sympathetic ganglia of the cases, but, undoubtedly, when these structures are involved in the general inflammatory reaction, they must suffer.

The participation of bloodvessels in the inflammatory process is of interest in connection with the symptom of intestinal hemorrhage. The tubercle itself is, of course, avascular but the granulation tissue, forming the floor and edges of the ulcer, is extremely

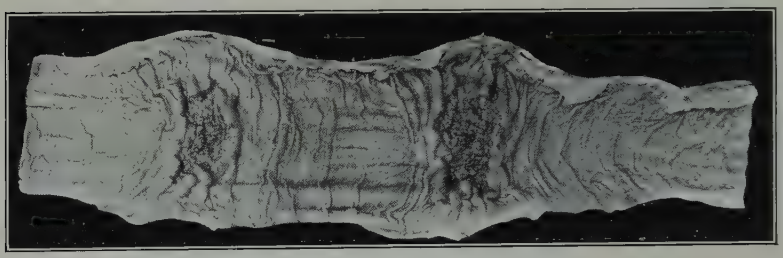

Fig. 12.—Annular ulcers in the ileum (Case 110).

rich in very large thin-walled bloodvessels. In no case has evidence of thrombosis been detected either in the newly-formed vessels or in those originally formed in the organ. The necrotizing process must frequently open many of these immature vessels and result in a

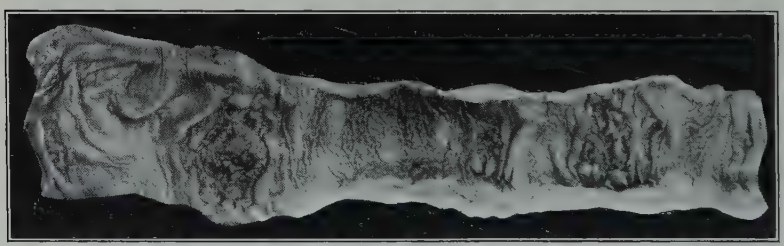

Fig. 13.—Annular ulcers in the ileum.

certain amount of hemorrhage. The more protected position of the larger arteries in the depths of the wall must explain the relative infrequency of massive intestinal hemorrhage.

INCIDENCE OF HEALING.

Thirteen or 40.6 per cent of the cases showed more or less evidence of healing. Of this number 11 had received artificial heliotherapy over periods of one month to four years; 2 had undergone colostomy or ileo-colostomy; and 1 had had no specific therapy. In 2 instances, where the lamp had been employed for four and six weeks respectively, no changes were noted which could be ascribed to treatment.

Healing in Untreated Cases. One case in this group requires particular mention. The patient had received artificial heliotherapy for only nineteen days, one month previous to her death, and yet at autopsy showed very marked evidence of healing in large numbers of the ulcers in the ileum. She had, however, what was considered a double infection, as, in addition to the healed or healing lesions in the ileum, there were interspersed between these, very numerous large active annular ulcers and in the colon a generalized more or less hypertrophic confluent ulceration. The degree of healing in the first group of lesions was too well marked to permit

FIG. 14.—Healed and active tuberculous ulcers—small intestine (Case 105).

its being ascribed to a short course of specific therapy so recently administered. Another patient in the untreated group, who died of acute generalized miliary tuberculosis, showed a small group of ulcers in the cecum with intensely inflamed borders. The gross appearance was that of an early process but the microscope demonstrated almost complete regeneration of the epithelium over a healing surface.

The histological features of healing in these two cases resemble in kind those to be described below in the group treated by the quartz mercury-vapor lamp. Essentially the process consists of a regeneration of the epithelium over areas of inflammation, which have

subsided and from which most of the specific tubercles have disappeared. In each of these cases, however, a few small slightly caseous tubercles remained in the angles of the ulcers to indicate the character of the original process.

Evidences of Healing in Cases Treated with the Quartz Mercury-vapor Lamp.—For purposes of description, the processes must be considered separately under various headings but it should be understood that all of them are progressing at the same time.

Regeneration of the epithelium, to a limited extent, can be observed in the intestine in all tuberculous processes which are not too active. Often in the *untreated* cases it was noted that the epithelium showed a tendency to regenerate, to creep around the overhanging lip of the undermined border of the ulcer and to cover its under surface. In none of these cases, however, has it been noted that any further growth of epithelium occurred.

In all cases *treated* for four months and longer, this epithelial regeneration has been much more extensive and in all the late cases it has completely covered the entire area of ulceration. It commences early, while inflammatory and destructive processes are still active, and often shows great irregularity.

In the small series of cases studied, regeneration has been observed only from the surface epithelium; there was no evidence of regeneration from the lower ends of Lieberkühn's glands, cut off in the process of undermining. Mitotic figures appear in the surface epithelium at the edges of the denuded area and with repeated division the newly-formed cells are pushed around the end of the lip of the ulcer, extend as a long attenuated strip of single flattened cells to the angle formed by the lip and the floor, and then turn back to cover the floor itself. If the latter is irregular they dip into its various folds and crannies. In the course of time the new lining cells assume a cuboidal or columnar shape with frequent accumulations of mucus-forming goblet cells. Later more or less irregular crypts of Lieberkühn develop. On the ulcer floor they are usually regular and typical but those, developing from that portion lining the under surface of the overhanging lip, grow upward into the substantia propria and between the normal crypts projecting down from the surface above. In the small tongues of mucous membrane and substantia propria, left between adjacent areas of ulceration, this new gland formation may be extremely complex.

In the untreated cases it was seen that, while the initial tubercle and its extensions by contiguity were frequently removed in the slough, the process continued to spread and undermine new areas.

In the treated cases extension of the disease is in some way limited. The mechanism is not suggested in any of the tissues yet studied. The early epithelialization of still active foci and the absence of great deformity in completely healed cases suggests that sloughing does not play a major role. In the cases studied, however, lymphatic and contiguous extension are limited, and in late cases the specific tubercles are confined to small proliferative lesions in the lymphoid tissue. The case under treatment for four years showed macroscopically scattered areas which were thought to be ulcers, healed by scar formation and many mammillations. The microscope, however, showed only thickening of the submucous connective tissue, which contained hyperplastic lymphoid tissue. The surface was completely covered by epithelium and no trace of tubercle could be

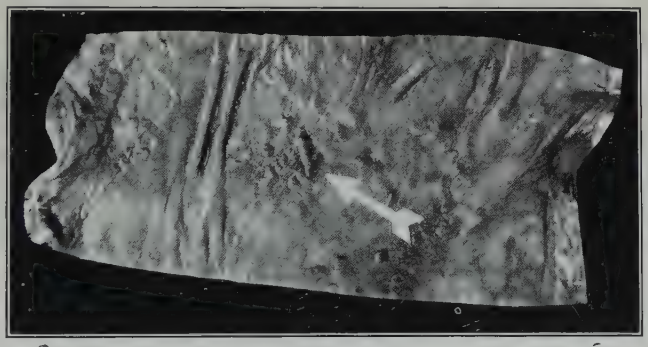

Fig. 15.—Mammillations (Case 129).

found. The scar formation was sufficient to preserve the outline of the original ulcer but its inflammatory character had completely disappeared. It is hoped, that as more cases are studied, the mechanism operative in preventing the spread of the disease will be suggested.

As already noted, tuberculosis in the intestine is characterized by extensive formation of perifocal granulation tissue, a reaction which is apparently accelerated and reinforced by heliotherapy. In the course of time, however, the granulation tissue is resolved as scar, and there results a more or less localized fibrosis in the submucosa, which compresses and invades the muscularis. This scar is non-cellular and contains a few large blood and lymphatic vessels. Contraction and stricture have never been observed. In the substantia propria, a limited amount of scar is developed in

the vicinity of irregular epithelial regeneration, and its thickening and contraction cause these areas to project from the surface as small mammillations. These bodies are macroscopic in size, from 1 to 3 mm. in length, and slightly less than 1 mm. in diameter. Thus far they have been observed in 4 cases treated for six months and longer. In them they occurred singly or in small groups. In gross there was no evidence of other lesion in the vicinity, but the microscope showed a moderate thickening of the submucous connective tissue, which would otherwise have been overlooked. In no instance could such formations be found in the vicinity of specific tubercle and, to the authors' knowledge, they have been described only in connection with healing tuberculous ulceration.

Postoperative Healing.—In this group there are only 2 cases coming to autopsy, respectively four and twelve months after their operations. The areas of macroscopic disease were infrequent and of course represented the remaining minimum after extensive resections. In each of these cases epithelialization had occurred but there were still persistent small caseous tubercles in the underlying tissues and some fibrosis. One case showed a definite stricture about an ulcer in the sigmoid.

In recapitulation, healing has been observed in 13 of 32 cases of intestinal tuberculosis. Of these, 7 were cases treated for a period of four months or more by radiation with the quartz mercury-vapor lamp. Two cases treated by this method for four and six weeks respectively, showed no evidence of healing. Healing was also observed in 2 cases treated by operation and in 3 without specific treatment. In those, who had had artificial heliotherapy, the healing was most effective in restoring the tissues to their normal appearance. This was particularly so in those cases where treatment had been continued for more than two and a half years. The completely healed lesions are characterized by a slight formation of scar tissue in the submucous connective tissue, complete disappearance of specific tubercle, lymphoid hyperplasia and the formation of mammillations projecting from the surface of the mucous membrane. It is apparently possible for almost complete *restitutio ad integrum* to occur, but very frequently death intervenes before this is accomplished. While the specific tubercles may have more or less completely disappeared from the picture, often the gross characteristics of extensive ulceration will still be preserved, in which case the microscope demonstrates an ulcer composed of granulation tissue covered by epithelium.

Evidence has been produced to demonstrate that healing may

occur without specifically directed treatment but these few cases would indicate the probability that radiation with the quartz mercury-vapor lamp initiates and accelerates the healing process. Observation of a much larger series of cases is obviously necessary before final conclusions can be drawn.

TABLE I.—TABULATION OF CASES SHOWING EVIDENCE OF HEALING.

No.	Extent of gross lesion.	Evidence of healing.	Duration of treatment.
105 20–3	*Small intestine.* Double infection, large active ulcers and small healing. Thinning of wall and dilatation throughout. *Large intestine.* Very active hypertrophic ulceration—cecum to rectum.	Very marked epithelialization and disappearance of tubercle; contraction of scar without deformity. Thinning of muscularis.	19 days only 1 month antemortem (not the cause of healing).
150 24–7	*Small intestine.* Small girdle ulcers. *Large intestine.* Large active ulcers, cecum to sigmoid.	0	1 month.
159 23 1	*Small intestine.* 10 to 12 ulcers duodenum. Many small round ulcers lower 30 cm. *Large intestine.* Ascending colon—scattered small ulcers, 1 in sigmoid.	0	6 weeks.
41 22 2	*Small intestine.* 2 microscopic tubercles. Areas of fibrosis in serosa. No pulmonary cavity.	Fibrosis of serosa.	2 to 3 months.
44 24–1	*Small intestine.* From jejunum to rectum. *Large intestine.* Many puckered often confluent ulcers with granulating bases.	Epithelialization over still active processes.	4 months.
147 21 5	*Small intestine.* Ileum 3 ulcers 4 mm. diameter. *Cecum*—1 puckered ulcer with surface adhesion.	Epithelialization. Shrinking of small non-caseous tubercles. Fibrosis.	6 months.
125 20–6	*Small intestine.* Lower 20 cm. 10 areas of roughening and radial scar granulations on overlying serosa. *Large intestine.* Very small ulcers valve, cecum and ascend-colon—very small ulcers lying in radial scars.	Fibrosis of tubercles; some persistent caseation. Epithelialization	6 months.
121 21 11	*Small intestine.* Mammillations. *Large intestine.* Cecum, papillary growth. Descending colon—mammillations.	Healed fibrous tubercles. Caseation in papillary growth. Fibrosis in cecum and lymphoid hyperplasia, mammillation.	6 months.

TABLE I.—TABULATION OF CASES SHOWING EVIDENCE OF HEALING.—(*Continued.*)

No.	Extent of gross lesion.	Evidence of healing.	Duration of treatment.
151 24–8	*Small intestine.* From duodenum to ileo-cecal valve, many longitudinal or round ulcers and small follicular ulcers—granulating floors. *Large intestine.* Involved to distal ⅓ of descending colon. Large confluent ulcers with red-brown base. Mammillations in descending colon. Large mass of caseous tubercle about cecum.	Epithelialization tubercle disappeared except 1 non-caseous in lymphoid tissue. Ulcers formed by very vascular granulation tissue. Mammillations.	1 year.
129 23–14	*Small intestine.* Lower ileum, areas of dilatation between foci where rugæ are puckered. No ulcer, mammillations. *Large intestine.* Descending colon and sigmoid. Areas of congested thickening. Slight constriction, mammillations.	1 small non-caseous tubercle, ileo-cecal valve. Scar in submucosa. Mammillation. No ulcer.	2¼ years more or less irregularly. Total period of actual radiation 10½ months.
158 24–9	*Small intestine.* A few small white scars visible on serosa of lower 20 cm. of ileum. Distortion of rugæ in interior. *Large intestine.* Very small ulcer ileo-cecal valve. Ascending colon, group very small ulcers, granulating bases. Sigmoid, puckered scar with deep narrow ulcer.	Epithelialization. Tubercle consists of a few cells in lymphoid tissue, and giant cell with a few endothelioid cells in submucosa. Mammillation. Scar in submucosa. Ulcers formed by vascular granulations. Sigmoid fibrosis.	4 years.
101 19–3	Cecum removed. Scar sigmoid.	No specific tubercle. Vascular granulation tissue. Fibrin on surface.	Death 3 months after ileo-colostomy.
135 21–1	Few small ulcers ileum and colon. Scar sigmoid.	Healing tubercles in lymphoid tissue. Ulcer with scar in sigmoid.	Death 1 year after ileo-colostomy.
22–5	Cecum. Multiple small inflamed ulcers.	Marked epithelialization caseous tubercles at angles of ulcers.	0
149 24–6	*Small intestine.* Few small ulcers terminal 45 cm. Marked lymphoid hyperplasia. Shallow inflamed ulcers surface ileo-cecal valve. *Large intestine.* Marked lymphoid hyperplasia and few small ulcers to middle transverse colon.	Healed tubercle in lymphoid tissue, which shows marked hyperplasia. Small lymphoid tissue ulcers. Thickened submucosa.	0

If the defects are not too great, says Rokitansky, the borders close together with their teeth forming a scar over the callous bottom to which the base has retracted and finally grow together. There always remains at the site of the ulcer a retraction of the wall, a fold, a ridge or teat-like projection, which forms on the inner surface of the bowel. In girdling ulcers this may form a projection ring.

So the healing, then, can take place not only by simple scar formation, but also in changing degrees of perfection, depending upon and by the formation of new epithelium. The muscular coats of course do not regenerate. Tubercle bacilli must disappear from the new tissue before healing can be considered complete. It is of great interest to note that this healing can occur or has been noted in certain ulcers, while the disease progresses in others or even in the same ulcer. The edges of such an advancing ulcer appear frayed or gnawed. The weakened condition of many patients, no doubt, prevents healing, though it can occur in advancing pulmonary tuberculosis, which is ultimately fatal. Healing, of course, is more likely to occur in the first two or three decades of life.

CHAPTER VII.

PATHOLOGICAL ANATOMY OF INTESTINAL TUBERCULOSIS (*Continued*). COMPLICATIONS AND SEQUELÆ.

STENOSIS.

Stenosis, which can occur either singly or, what is more common, multiply, is found more frequently in the small bowel, though also in the large. It is infrequent in secondary intestinal tuberculosis as death usually occurs before healing sufficient to produce it takes place. In 1 case between the cecum and the duodenum twenty strictures occurred and in 1 between two stenoses were calculi the size of a shot (Gayet). The stenosis, while usually not very marked, may be so pronounced that only a very small sound can pass through it. Above the stenosis the bowel may be dilated and at times form a large pouch, and the walls thickened, due in part to hypertrophy of the muscular coat.

The cause of stenosis is thought, and with much reason, to be the result most frequently of cicatrization due to more or less imperfect healing of previous ulceration. On the other hand, purely spastic strictures are said to occur at times in the neighborhood of tuberculous lesions. Again, the view has been advanced that tuberculous granulation tissue, due to tubercle bacilli of low virulence, can girdle the intestine without producing ulceration, and, on contraction, produce stenosis. Some claim on the other hand that stenosis is never due to a non-ulcerative form of intestinal tuberculosis and that the fact, that the constricted area is covered with epithelium, proves nothing, for the original ulcerative process may have long preceded the examination and, furthermore, that the inclusion of epithelial nests in the scar is proof that the process was originally ulcerative. Beside the shrinking and pulling of the scar, there is present a faulty action of the musculature, by which the circular muscles, due to the breaks in the longitudinal muscular layer, produce an invagination of the serosa. It is possible that these strictures are not due solely and always to ulceration, to the tubercle bacilli and to the inflammation and caseation it causes, but also to

the partial or complete obliteration of the bloodvessels supplying the diseased area (O. Busse). Cornil reports a case where stenosis was due to the tuberculous condition of two opposing valvulæ conniventes.

Bernay and later Nikoljski have described a rare form of stenosis, due to a primary ulcer in the serosa with a thickened wall but a normal mucosa.

Hofmeister held that multiple strictures are due to the cicatrization of the usual annular ulcers, while the single strictures are often caused by the hypertrophic form.

Considering the frequency of tuberculous ulcerations in the bowels, especially in pulmonary tuberculosis, and the rarity of stenosis, certainly of a clinical type, probability is lent to the view that stenosis more frequently follows primary than secondary intestinal tuberculosis, where the pulmonary lesion frequently causes death before healing can advance far enough to produce stenosis.

PERFORATION.

Perforation of a tuberculous intestinal ulcer is not uncommon. If we assume that 75 per cent of all cases of chronic pulmonary tuberculosis have intestinal ulceration and that 2.5 per cent of all cases of chronic pulmonary tuberculosis have perforation, complete or incomplete at death, it would then appear that 3.7 per cent of all cases with intestinal ulcers had perforation. If, on the other hand, we assume that nearly every case of typhoid fever has intestinal ulceration, that 20 per cent (high for the sake of argument) die, and only 10 per cent of these from perforation, then it can be seen that, while only 2 per cent of cases with typhoid ulcers have perforation, 3.7 per cent of cases with tuberculous ulcers suffer perforation. In any case, it would appear that perforation is at least as common in intestinal tuberculosis as in typhoid fever. The actual percentages found at autopsy vary from 1.2 per cent in 2000 autopsies on pulmonary tuberculosis reported by Fenwick to 5 per cent by Willigk. Louis's and Dittrich's results agree with those of Fenwick. Lebert and Schwatt found 3 per cent. Again, Habershon found in 56 cases of acute peritonitis following perforation 4 (7 per cent) to be due to ruptured tuberculous ulcers, while Grawitz found 20 (2.3 per cent) in 867 cases, Osler 13 (2.7 per cent) in 475 autopsies, S. West 2 (0.6 per cent) in 326 cases and Girode 2 (7 per cent) in 29 cases. The ileum is the usual seat of the perforation but is followed closely by the cecum.

In comparison with the large number of lesions perforation is rather infrequent which is due: (1) To the fact that as the ulcers deepen the bases are thickened by inflammatory processes and by fresh and closely aggregated tubercles; (2) to the occurrence of a localized adhesive peritonitis, matting together loops of the intestine and the omentum; and, (3) to the fact that death supervenes from pulmonary tuberculosis before the ulceration extends through the wall (Schwatt).

The cause of the perforation may be mechanical and due to increased pressure which occurs, for example, just above the ileocecal sphincter or above a stenosis, or to a severe acute ulcerative process. In 1 instance a roundworm was found to have burrowed through the base of a tuberculous ulcer and caused fatal peritonitis.

Perforation occurs most often only in patients with extensive ulceration, though Schwatt questions this, and possibly in those who for some reason have not received the best of care. It may occur, however, in the only ulcer present.

Perforation may occur into the free peritoneal cavity or, as occurs more frequently, into another viscus or into a mass of adhesions. Rupture through the skin, into other coils of the intestine, into the bladder, pleura, Fallopian tube and uterus, have been described. In 1 instance successive ruptures occurred until the food passed from the duodenum into the cecum, never entering the jejunum or the ileum.

The results of perforation may be either general peritonitis (rupture into the free peritoneal cavity), or fecal abscess.

PERITONITIS.

Fenwick, in an excellent study, discusses 15 cases of his own and 28 cases collected elsewhere of acute general peritonitis. He analyzed 34 cases and found 28 with a single ulcer which had ruptured into the peritoneum. Males were more affected than females (2 to 1). In 27 instances the small gut was involved (duodenum 3, jejunum 3, ileum 21), and in 7 the large (appendix 4, cecum, ascending and transverse colon, 1 each). In a series of 77 cases, W. Fox found 3 cases of perforation, all in the large bowel. In 226 collected cases of tuberculous peritonitis Borschke found the bowel tuberculous in 86. Schwatt found serofibrinous or purulent peritonitis in 7 of his 134 cases.

Localized peritonitis is usually found covering the areas of the ulcers, whether superficial or deep, though the serosa may be normal

in appearance or covered with white tubercles. Gardner and Letulle have both noted this localized peritonitis, or serositis, as it might be called.

FECAL ABSCESS.

Fecal abscess is more frequent than general peritonitis. In 122 cases of fecal abscess, Fenwick found that only 10 came from perforation of tuberculous ulcers. He discovered these in the 2000 autopsies he had studied and collected 15 others. It occurred more frequently in females (3 to 2), usually between the ages of twenty and thirty years. The sack was found in the peritoneum or even in the muscle. It occurred more frequently in rupture of the large intestine than of the small bowel (3 to 1). In 10, the abscess was in the right iliac fossa, in 5 in the center of the abdomen, in 3 it was superficial. In 6 the perforation was in the cecum, in 3 in the ileum, in 2 in the appendix, in 1 in the jejunum and in 1 in both duodenum and cecum. In 12 the abscess burst during life, once into the peritoneum, 4 times in the groin and 3 times into the bowel.

Senckenberg and his coworkers, from their observations in the pathological institute at Frankfurt, hold that large quantities of tubercle bacilli gain entrance from the intestinal lesions into the portal circulation and thoracic duct, greatly influencing the course of the pulmonary lesions, and, at times, causing general miliary tuberculosis. The frequent infection of the liver in such conditions is probably due also to this reason.

Fischer found microscopic tubercles in the liver of 30 patients coming to autopsy, who had tuberculous ulcers of the bowel, and thinks they practically always occur under such conditions. Zehden found miliary hepatic tubercles in 50 per cent of all fatal cases of pulmonary tuberculosis. Rolleston thinks these figures are too high.

Carcinomatous degeneration has been found in the tuberculous ulcers (see p. 225).

CHAPTER VIII.

PATHOLOGICAL PHYSIOLOGY OF MOVEMENT.

The pathological physiology of the intestine is of great interest. Much work has been done and much remains still unsolved. As the depth of the tuberculous ulcers varies very greatly, as we have seen, it is conceivable that in some cases only the epithelial layer may be lost over the tuberculous lesion in the lymph follicle. Again in others only the peritoneal coat may be left. Many ulcers must penetrate to depths between these two extremes. About the ulcers must be some inflammatory change which in turn must affect all the structure of the bowel.

In intestinal tuberculosis hypermotility and spasm are very frequent. The former is shown by diarrhea and by rapid emptying of the bowel. How is this brought about? It is probable that several factors are at work. As already mentioned some studies have shown that not only has the bowel been subjected to ulceration in intestinal tuberculosis, but also that a widespread inflammation involving a considerable area about each ulcer and affecting the myenteric plexus and the muscular coats is present. The muscle under these conditions becomes more irritable. Segments from the intestine of a rabbit, just above a badly inflamed Peyer's patch, contracted twice as rapidly in aërated Locke's solution, as it should normally have done (Alvarez). It may also increase the tone (spasm) as is seen frequently in ulcerated areas of the intestine. This is of especial interest as the tuberculous poisons usually depress muscular tone and activity. The muscle of the ileum is said to be less sensitive to adverse conditions than that of the duodenum and the colonic muscle less so than either.

Auerbach's plexus, thus, does not escape this inflammatory process in intestinal tuberculosis. In some ulcers not only is Meissner's plexus but also Auerbach's plexus involved in the ulceration and destroyed, but it is much more unusual to find the latter so affected. Consequently, the motor nervous mechanism of the intestine is more rarely completely deranged and, while the endings in the mucosa and submucosa over the ulcers may be destroyed, the nerve impulses may be conducted as well as, or even better, than in normal tissue, and so bring about increased peristalsis.

Attention must again be drawn to Leupold's work on the involvement of Auerbach's plexus in intestinal tuberculosis. It will be recalled that, in cases with diarrhea, degeneration of the ganglion cells and round-cell infiltration were much more widely spread, and inflammation of the nerve sheaths (Askanazy) was present. Such conditions, no doubt, play a rather important part in the motor changes resulting in diarrhea.

Unquestionably, the number and location of the ulcers play some part in the increased movement of the intestines (*i. e.*, diarrhea). When the ulceration is confined to the small intestine, constipation with or without diarrhea may occur, but ulceration in the ascending colon leads usually, and, when in the descending colon, nearly always to diarrhea. Marked ulceration at the hepatic flexure, however, may be accompanied with constipation.

A patient, who had suffered from diarrhea for ten weeks, had for five weeks before death only one loose stool a day. At autopsy the entire large bowel was studded with innumerable ulcers, so extensive, in places, that only islands of mucous membrane were left (Kortum). It is possible that in this case the reflex arcs were destroyed. It is of interest to note that N. Moore found the whole surface of the large bowel involved in tuberculous ulceration in a patient who had no diarrhea. Austin Flint, in 10 autopsies on patients who had had persistent and permanent diarrhea, found in 1 case only one ulcer just above the cecum; in another, a few small ulcers without thickening of the wall or inflammation about them.

Some have attributed the diarrhea to the poisons in the swallowed sputum, acting possibly on a sensitized neuromuscular system. Poor absorption on account of blocked lymphatics, due to enlarged mesenteric glands, often tuberculous, or to other reasons, may have some influence on the contents of the small, but little on that of the large bowel, where the absorption of water usually occurs. A catarrhal condition of the mucous membrane, lying between the ulcers, widespread infiltration and irritation of the nerve plexus, amyloid degeneration, may all act to cause diarrhea in different cases. The deep ulcers, some have suggested, paralyze the muscular coat.

The pain, so frequent in intestinal tuberculosis, can be explained by the increased tone of the muscle. The law of the intestine, *i. e.*, relaxation of a distal, before contraction of a proximal portion of an intestinal segment (Meltzer's "law of contrary innervation"), therefore, cannot take place, as the ulcerated segment is often in a

state of spasm or, indeed, cannot relax on account of pathological changes. The descending wave of contraction, forcing the intestinal contents (gas or fluid) before it, causes such increase of tension (or distention) that colicky pain results, due to pressure on the nerves, which, of course, stenosis and hypertrophy of the muscle would increase. It is probable that, only when there is abnormal motor activity, does pain occur. In the absence of diarrhea, obstruction and localized peritonitis or serositis, intestinal ulceration may cause no pain. The contact of the intestinal contents with an ulcer causes no pain. The pain, occurring during or after meals, may be explained by the fact that the consumption of food and its passage through the pylorus and duodenum cause ileal and colonic contractions. The intestine throughout (*i. e.*, to the anus) is insensitive to tactile and thermal stimulation, and the surfaces of intestinal ulcers are no more sensitive to tactile and chemical stimuli than other portions of the bowel. It should never be forgotten that the intestine is poorly supplied with sensory nerves.

The loss of appetite, disgust for food, nausea at the sight or smell of food, a feeling of fullness after a few mouthfuls may be due to what Alvarez terms a reverse gradient which is really reverse peristalsis.

CHAPTER IX.

THE ETIOLOGY OF INTESTINAL TUBERCULOSIS.

Ulcerative intestinal tuberculosis can arise from infection either by way of the blood or lymph, by direct extension or, what is most frequent, by direct contact.

INTESTINAL TUBERCULOSIS BY DIRECT CONTACT.

The cause of intestinal tuberculosis, secondary to pulmonary tuberculosis, has long been accepted as due, practically always, to the tubercle bacilli in the sputum, swallowed consciously or unconsciously, which was first suggested by Edwin Klebs. From time to time, this has been questioned but many points have been marshaled in its favor. Attention has been called to the fact that the most frequent secondary tuberculous complications in pulmonary tuberculosis occur in those parts constantly undergoing, while functioning, more or less stretching, and over which tubercle bacilli, if present in the sputum, constantly pass, *i. e.*, the larynx and the intestine.

Number of Tubercle Bacilli.—Some autopsy studies seem to indicate that intestinal tuberculosis occurs more frequently in those patients with large numbers of tubercle bacilli in the sputum. Now, tubercle bacilli apparently occur somewhat more often in large numbers in the sputum of patients with pulmonary vomica than in those without. In a study of 277 of our cases, pulmonary cavities were absent in 70 per cent of 146 cases, in a moderately advanced stage, and present in 73 per cent of 131, in a far advanced stage. Among the cases without pulmonary cavities, 69 per cent, with sufficient data about the number of tubercle bacilli, fell in Gaffky VI or below, while of those with pulmonary cavity, as many were over Gaffky VI as were Gaffky VI or under. (Gaffky VI indicates 7 to 12 tubercle bacilli to the field). Eisenhardt found cavities in the lungs of 489 (87 per cent) of 563 persons dead with pulmonary and intestinal tuberculosis, and some would even increase this large percentage. In 108 cases of pulmonary tuberculosis with cavities, Edens found 91 had also intestinal tuberculosis. In 277 of our

cases, in which accurate data could be obtained, pulmonary cavities, determined by roentgen-ray study, were present in 47 per cent. Gardner found pulmonary cavities in 29 of his 32 cases and, in 2 of these without cavities, found only microscopic evidence of intestinal tuberculosis.

It has been stated that the insane with pulmonary tuberculosis are more prone to intestinal tuberculosis on account of swallowing their sputum, a fact not yet sufficiently substantiated.

Children, on the other hand, have secondary intestinal tuberculosis much more rarely than adults, and, while they swallow their sputum, they also rarely exhibit cavities in their lungs or open tuberculous lesions. Again, it must not be forgotten that tubercle bacilli may pass through the intestinal wall more easily in children, and that the lymph glands have also wider spaces. Kieffer found 347 (77.3 per cent) of 462 patients over forty years of age and 95 (58.6 per cent) of 161 patients under forty years of age, had intestinal tuberculosis, the diagnosis being based, presumably in part, on clinical data and not on actual autopsy findings. Bollinger found in children with pulmonary tuberculosis only 30 to 40 per cent with intestinal tuberculosis. Biedert collected 1346 cases of tuberculous children, who at autopsy showed involvement of the intestine in 31.6 per cent. The same thing is true in animal tuberculosis. Cornet noted intestinal tuberculosis only 6 or 8 times in 4000 examinations of tuberculous animals, and it is exceedingly difficult to produce ulcerative intestinal tuberculosis experimentally.

It is unquestionably true that intestinal tuberculosis occurs more frequently in patients with large numbers of tubercle bacilli in the sputum, but whether this is the cause of the intestinal tuberculosis, or whether larger numbers of tubercle bacilli occur in the sputum as the patient is losing his power of resistance and is more likely to develop or, indeed, has already developed this complication, is not yet decided. Cornet suggests that, as long as a patient can eat food enough to dilute his swallowed sputum, he is less likely to have intestinal tuberculosis, but here, again, it is not easy to distinguish cause and effect. Swallowing bacilli, when there is no food in the stomach, is probably more dangerous.

Again, it must not be forgotten that swallowed sputum (pyococci) can apparently set up a catarrhal condition of the intestinal mucous membrane. This catarrhal inflammation, with epithelial desquamation and formation of erosions, possibly a pretuberculous enteritis, occurs, some hold, frequently and favors penetration of the tubercle bacilli according to Hanot, Rilliet and Barthez and Rolleston.

Benoit thinks that abnormal ferments enfeeble and irritate the mucosa and so lead to or cause extension of the disease.

More or less recent work has demonstrated that even in the saliva of some patients tubercle bacilli can be found. It has also been shown that tubercle bacilli occur in the stools of practically all patients, who have them in the sputum, and in some they can be discovered apparently only in the feces and not in the sputum. Tubercle bacilli may enter the intestine through the bile, as L. Rabinowitsch, Fraenkel and Krause, Calmette and Corper have demonstrated, but, as Schöne and Weissenfels have shown, the numbers from this source are so small that they can be largely disregarded, although Sasano and Medlar affirm that intestinal tuberculosis in guinea-pigs arises in this way.

After weighing all the evidence, it seems highly probable that the number of tubercle bacilli present in the sputum and, consequently, in the feces, is less important than the prolonged contact of tubercle bacilli with the mucous membrane of the bowel. In other words, prolonged contact of the bacilli with the mucous membrane, whether in large or small numbers, is the most important etiological factor in the production of secondary intestinal tuberculosis.

Does intestinal tuberculosis precede, follow, or occur simultaneously with the formation of pulmonary cavities? The problem has not yet been worked out, but some facts might lead us to hold that the process of cavity formation in lung and bowel wall may go on simultaneously, and is due to some change in reaction capacity which occurs throughout the body.

Again bearing in mind Römer's work on the production of pulmonary cavities in experimental animals, it would appear possible that, not until large numbers of tubercle bacilli are absorbed through the ulcerated areas in the intestines, gain entrance to the venous blood and finally reach the lungs, would pulmonary cavity formation proceed in some patients. Tubercle bacilli we know are constantly passing through the walls of the intact and normal intestines. In experimental animals this has been proved by Ravenal and others. The occurrence of tuberculosis of the mesenteric glands in patients with pulmonary tuberculosis may be assumed as presumptive evidence that the glandular infection has occurred through the intestinal wall, which may be intact. The difficulty of producing intestinal tuberculosis in animals, even when emulsions containing enormous quantities of tubercle bacilli are dropped down the esophagus, and the great frequency of intestinal tuberculosis in advanced pulmonary tuberculosis in man, would point to the fact

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that the mere presence of tubercle bacilli in the intestinal contents, or even in the intestinal wall, is not sufficient to produce ulcerative intestinal tuberculosis. The explanation may lie in the fact, that in pulmonary tuberculosis the swallowed sputum, overfeeding, indiscretions in diet and other causes may set up a catarrhal condition of the mucous membrane and so favor entry and implantation of the tubercle bacilli, or, again, it may be that certain allergic or anergic states are more likely to occur from time to time in patients with chronic pulmonary tuberculosis, whose disease extends over many years, and who have tubercle bacilli constantly in the intestinal contents and wall, and suffer, from time to time, from other complications. In any case, it appears that mere contact of the intestinal wall with the tubercle bacilli is not sufficient to produce intestinal tuberculosis, either in man or in animals.

Action of Gastric Contents.—In passing, it may be of interest to recall that the stomach is rarely affected with tuberculosis. This is due, some think, to the fact that the acid secretions in the stomach have little effect in dissolving the sputum, which, on the other hand, is readily dissolved in the alkaline intestinal contents. This, of course, would bring more tubercle bacilli in contact with the intestinal than with the gastric mucosa and may explain the immunity of the duodenum. It has been suggested that the frequency of tuberculosis in the ileo-cecal region may be due to the attenuating effect of the hydrochloric acid or of ferments in the stomach upon the tubercle bacilli, which recover their usual virulence only when they reach the ileo-cecal sphincter. As this requires but one to three hours, it is probably of little moment.

The action of the gastric juice of a young healthy dog had no apparent effect on the virulence of a pure culture of tubercle bacilli at the end of six, but did destroy them after twenty-four and forty-eight hours (I. Straus and Wurtz). Macfadyen found that tubercle bacilli were affected only by strengths of pepsin and HCl that never occur in the stomach. E. Frank obtained practically the same results. Wesener found that the "spores" of the tubercle bacilli were left intact but that possibly their growth was inhibited. Inkster and Gloyne withdrew with an Einhorn tube the gastric contents after a meal of oatmeal. They incubated tubercle bacilli as long as two hours with the gastric juice thus obtained, and always produced tuberculosis in guinea-pigs. Some tubercle bacilli they thought might be killed.

The real problem in etiology is, of course, not whether tubercle bacilli can get into the intestinal wall, which they do readily either

mechanically (Baumgarten) or in wandering cells or leukocytes (Tchistovitch), but what mechanism brings about their lodgment and proliferation there.

Site of Lesions.—The most frequent site of intestinal tuberculosis is in the ileo-cecal region. Many explanations have been offered. The digestive processes in the alimentary tract are completed in the terminal ileum. Absorption of the last of the products of digestion takes place here. The meal is longer in contact with the ileal walls than with those of the stomach. The lymph masses of the intestinal walls (solitary follicles and Peyer's patches) are most numerous here. Tuberculous infection today is considered to originate most frequently in lymphoid tissue. Consequently, it would be quite natural to conclude, that, where the food and sputum have reached their greatest degree of digestion, where absorption is very active, where the products of digestion and undigestible residue linger longest, where the greatest amount of lymphoid tissue occurs, is where intestinal tuberculosis would most likely occur.

Upholding this view in part is Hanau's observation on 3 cases of intestinal tuberculosis, all of which had tuberculous cavities in their lungs. In 1, there were no Peyer's patches and no tuberculosis of the small bowel, but ulcerative colonic tuberculosis. In a second, the Peyer's patches were very small and no tuberculosis was found, except on the ileo-cecal valve and in the colon. In a third, where there were many well formed solitary follicles, but only one Peyer's patch, many of the follicles were swollen and caseous, and two small ulcers occurred. In the ascending colon were many ulcers, but fewer in the transverse colon.

Next to the terminal ileum, the cecum is probably most often affected. Here again the contents lie long in contact with the walls. Here the brunt of the reversed peristalsis when it does occur must be borne. If prolonged contact is of moment as a causative factor, patients with marked constipation should be more prone to develop cecal tuberculosis. On the other hand, prolonged rest of the feces in the large bowel may lead to marked inspissation and so lessen the chances of contact of the tubercle bacilli with the walls. It is possible, of course, that each ulcer increases the chance of infection for the more distal parts, for tubercle bacilli coming from the ulcers would come in contact with the lower segments of the bowel. It is of interest to note, however, that the tuberculosis decreases in frequency in direct relation to the distance from the ileo-cecal sphincter.

It is not easy to explain by this view the tuberculosis of the jejunum and higher ileum, but breaks in the continuity of the mucous

membrane may play a part. Secondary ulceration which not infrequently occurs above a stenosis may be brought about by prolonged contact of the contents with the wall at this point and, possibly, by mechanical injury as well.

HEMATOGENOUS INFECTION.

Infection of the intestine by means of the blood and the lymph can undoubtedly occur. Miliary tuberculosis of the whole intestine, occasionally with ulceration, while rare, is certainly most likely a blood-borne infection. It is not very difficult, on the other hand, to conceive how the mucous membrane of the whole tract could be so changed as to render it simultaneously susceptible to enterogenous infection, for, in some of the cases, the lesions are apparently all of the same age. It is of interest to note that in 100 cases of intestinal tuberculosis, Engelsmann found the larynx affected 67 times, and Sahlgren 7 times in 40 cases which, of course, suggests lessened resistance. On the other hand, some (Archibald, Calmette, Baumgarten, Orth) would have us believe that the hematogenous is the usual mode of infection, in disease secondary to pulmonary tuberculosis. It has long been known that tubercle bacilli can pass through the normal, intact intestinal mucous membrane. How this occurs has never been definitely determined. Lacunæ between cells have been described in young animals, but nearly always leukocytes are found lying between the epithelial cells and it is easy to conceive how such cells, on the edge of the mucous membrane, could phagocyte tubercle bacilli and wander into the lymph or blood stream.

Tuberculosis of the liver, often found only microscopically, is, according to some, a nearly constant occurrence in intestinal tuberculosis, due to tubercle bacilli which have gained entrance into the portal vein. Some observers, as already mentioned, would lead us to believe that tubercle bacilli, absorbed in this way through the lymphatics, would pass through the lungs and finally find lodgment in the lymphoid tissue of the bowels. Such a process seems unlikely. It is of interest to note here that Fischer and others believe that tubercle bacilli gain a ready entrance into the blood stream through the tuberculous ulcer.

Baumgarten as well as Loeper and Esmonet, produced intestinal tuberculosis experimentally by intravenous injection (into the mesenteric artery). Betke reports a case, a girl, aged four years, where a caseous gland ruptured into a pulmonary vein, causing meningitis and death. At autopsy, tuberculous ulcers were found in the bowel.

Engelsmann quotes this as a certain proof of hematogenous infection, but it is questionable whether the intestinal tuberculosis was not the primary infection.

In a recent and suggestive article, Lenoble states that intestinal tuberculosis arises from hematogenous infection. The tubercle bacilli reach the intestinal wall by the mesenteric arteries in which are found emboli. No tubercle bacilli, he states, are found in the lumen but many in the arterial or capillary wall. The tubercle bacilli pass by diapedesis to the surrounding tissue, and may be found at first at a distance from the mucosa and from the glandular tissue. The follicles are next invaded; the tubercle bacilli multiply; coagulation necrosis supervenes; and the usual picture of intestinal tuberculosis is soon laid bare. Finally, tubercle bacilli pass into the lymphatics and so to the mesenteric glands and liver. The ulceration is caused by other bacteria or massed numbers of tubercle bacilli. It would appear that Lenoble has reversed the sequence of events. Gardner noted no emboli or thrombi in his studies.

Sasano and Medlar believe their experimental work in guinea-pigs and a study of one human case demonstrates that intestinal tuberculosis arises from infection from the intestinal contents. Gardner believes that, where isolated groups of lesions approximately all of the same size occur high up in the intestine, hematogenous infection may have occurred, but, in 2 of his 32 cases, where such lesions occurred, no evidence of such involvement could be found microscopically.

Hematogenous infection, then, while possible, must be very rare.

The intestine can of course be involved in tuberculosis by direct contact with a contiguous organ or from the peritoneum.

It is of interest to note that in man bovine tubercle bacilli apparently cause intestinal tuberculosis less frequently than the human type. This may depend not upon any difference in the two organisms but rather upon the different pathological reactions of the host. It is apparently more frequently the cause of primary intestinal tuberculosis than the human type.

Bowel disturbances, such as diarrhea and constipation as well as previous ulceration (*e. g.*, in typhoid fever), are considered at times important etiologic factors. Birch-Hirschfeld, Errot, Hansemann, and others have found tubercles in typhoid ulcers.

Von Behring held that the mucus on the surface of the intestine protects it from attacks by the tubercle bacilli. This was lacking in the young. Disse says this does not hold for animals as a mucous layer overlies the epithelium at all ages.

Pottenger calls attention to the possibility of trophic disturbances in favoring implantation. "The nerves (both vagus and sympathetic) which supply the intestinal tract suffer a prolonged central stimulation from toxins produced by and as a result of the disease, as well as peripheral irritation by the inflammation in the lung, which must necessarily cause trophic changes in the cells of the intestinal wall, thus lowering their resistance to infection."

The etiology of intestinal tuberculosis will never be fully cleared up until a method of producing it experimentally in laboratory animals has been devised.

CHAPTER X.

EXPERIMENTAL INTESTINAL TUBERCULOSIS.

The production of experimental ulcerative tuberculosis in the small laboratory animals is very difficult. Villemin (1868), after feeding tuberculous tissue suspended in water to rabbits, found tuberculous ulcers in the small gut and cecum in some, a few tubercles on the aggregate follicles in others, while a certain number showed no intestinal lesions. In another experiment, he fed tuberculous sputum (40 gm.) to guinea-pigs. At the end of three months, they died or were killed and the small bowel and cecum were found affected.

Klebs, who began in 1868 his feeding experiments, in 1870, fed guinea-pigs tuberculous matter and found tuberculous ulceration of the intestines in 2 cases. He found it easier to produce hyperplasia of the follicles and intestinal ulceration by feeding caseous and fresh tuberculous tissue to herbivora (sheep and goats) than to carnivora.

Chauveau, however, in 1869, obtained 4 heifers (aged six to twelve months) from the mountains as he thought they would more likely be free from any tuberculous taint. One was used as a control and the other 3 were each given, in water, 30 gm. of tuberculous tissue from an old tuberculous cow. After fifty-three days, 2, very ill and cachectic, were killed and their intestines, especially the ileum, were found sprinkled with tuberculous ulcers. In a second experiment (1872), he fed 11 cattle, aged fourteen months, bovine and human tuberculous organs, some in large quantities for four or five days, others only once. Many developed intense diarrhea and some showed slight, others overwhelming intestinal infection in the solitary and aggregate follicles. In a third experiment, he failed to produce intestinal lesions in 2 animals fed on milk. He verified Villemin's work and attributed much importance to intestinal infection.

Gerlach, in 1870, in a series of experiments, found gray miliary tubercles in the intestines of 2 rabbits.

Liesering, in 1872, fed 2 sheep tuberculous lymph glands from a cow and at autopsy, on the seventy-eighth and eighty-third days, found tuberculous ulcers in the small bowel.

Zürn, in 1872, found single ulcers in the intestines of a pig, fed with the tuberculous liver of a cow.

Bollinger (1873) fed 2 goats 20 gm. of a tuberculous lung and, while they died in fifty-one and sixty-five days, neither had intestinal tuberculosis.

Viseur, in 1874, fed tuberculous lungs from cattle to 5 cats and 4, which were young, developed caseous ulcers in the solitary and aggregate follicles, but the old cat escaped. In a second experiment on 3 dogs and 4 cats, killed after two months, the 4 cats and only 1 dog had intestinal tuberculosis.

Orth (1879) fed 15 rabbits with bovine tuberculous tissue and 9 developed tuberculosis of the intestines and other organs. He says one must wait six months to get advanced intestinal tuberculosis.

Blumberg, in 1879, by feeding tubercle bacilli in sputum and portions of human lungs to sheep obtained many swollen but no ulcerated follicles and Peyer's patches.

Toussaint (1880) fed 2 young pigs a whole lung from a tuberculous cow. One was killed in seventy-seven days and 1 died at one hundred and one days, and both had intestinal tuberculosis.

Peuch (1880) produced diarrhea in a rabbit, dying one hundred and thirty days after ingesting one-quarter liter of milk from a tuberculous cow. Many tuberculous granulations, especially in the ileum, and ulcers of the Peyer's patches were found.

Two of 8 sheep, fed tuberculous bovine tissue by Siedamgrotsky (1882), had intestinal tuberculosis at the end of three to six months.

Baumgarten (1884) found tuberculous ulcers in the small and large gut in rabbits, fed with tuberculous organs of rabbits and guinea-pigs, suspended in 50 to 100 gm. of milk.

Wesener (1885) was able to produce intestinal tuberculosis in rabbits repeatedly by feeding tuberculous sputum (nearly always mixed with sodium bicarbonate), and also, in 1 case, by subcutaneous injection.

Fischer, in 1886, fed rabbits milk with tuberculous material (some of it decomposed) from rabbits' lungs and got typical intestinal tuberculosis among other lesions.

Dobroklonski, in 1890, gave guinea-pigs a pure culture of tubercle bacilli in the mouth and found that after ten days some isolated follicles of the Peyer's patches began to swell and to project into the lumen of the gut. The vessels in and about them were dilated and had about them an increase of lymphoid cells. From then on the process advanced rapidly. Soon similar inflammatory foci formed outside of the Peyer's patches. About the twentieth day, microscopic miliary tubercles were found in the solitary and aggre-

gate follicles, occurring rarely just under the epithelium. These changes were confined almost wholly to the ileum and cecum. On the fourth day, he found tubercle bacilli in the mesenteric glands and in the intestinal wall, as well as upon the epithelium. In a review of 100 preparations, he often found tubercle bacilli and frequent nodules in the tissue of the villi, in part at a certain distance from the intact epithelial layer, and, in part, just under it. So tubercle bacilli can pass through normal intestinal mucous membrane.

Uffenheimer injected tubercle bacilli into the rectum of animals and found only the descending colon and rectum involved.

Van der Sluys found bits of bone mixed with tuberculous organs aided in infection.

Medlar and Sasano inoculated 8 guinea-pigs subcutaneously with 20,000 tubercle bacilli (H 37). One had macroscopic and 6 of the others microscopic intestinal tuberculosis. All had hepatic lymph node and mesenteric lymph-node tuberculosis. The lesion starts as an inflammatory exudate of polymorphonuclear leukocytes in the lumen of the glands, dipping down into the lymphoid tissue. The tubercle bacilli are chiefly in the polymorphonuclear cells, which undoubtedly carry them into the submucosa. The majority of the mucosal glands show no inflammatory reaction within their lumen. Tubercle bacilli occur only where there is inflammatory reaction. In 1 human case the findings were similar. The mesenteric glandular and hepatic tuberculosis paralleled the intestinal tuberculosis. If tuberculous peritonitis is excluded, they continue, intestinal tuberculosis is always the cause of the mesenteric lymph node tuberculosis. Intestinal tuberculosis in the guinea-pig is produced by the tubercle bacilli excreted in the bile, for no evidence of hematogenous infection was found.

I. Straus concludes that intestinal ulceration is far from being constant in animal experimentation; that, in feeding experiments, the lymphoid tissue of the intestine (solitary and aggregate follicles) is first and most markedly affected: that intestinal tuberculosis is easier to produce in young animals; that it is easier to produce it in ruminants (cattle, sheep, goats) than in carnivora (cats, dogs), easy in gnawers (rabbits, guinea-pigs), and relatively easy in pigs.

Calmette is convinced that intestinal tuberculosis arises from a blood-borne infection, usually secondary, and can be produced in animals by an intravenous injection. Margarucci, and Loeper and Esmonet have produced true tuberculomata by injecting tubercle bacilli into the intestinal arteries of dogs. At times similar lesions occur in guinea-pigs after subcutaneous inoculation.

CHAPTER XI.

THE IMPORTANCE AND FREQUENCY OF SECONDARY INTESTINAL TUBERCULOSIS.

If intestinal tuberculosis were only a terminal complication, it would be wise to maintain the attitude held for many years, viz., that after a diagnosis of intestinal tuberculosis, if the patient died, the diagnosis was correct; if he recovered, the diagnosis was wrong. It is true that in the minimal or incipient stage only a few cases occur. At the Trudeau Sanatorium which accepts only favorable and usually early cases of pulmonary tuberculosis, a study of 361 routine cases showed that 23 were positive, of which 18 had moderately advanced and 5 far advanced pulmonary tuberculosis. There were no cases of intestinal tuberculosis among the minimal group. This whole group embraced 65 minimal, 221 moderately advanced, 41 far advanced cases and 34 suspected and non-tuberculous cases.

It is possible that some of these cases may have had intestinal tuberculosis early in their disease. It is only fair to state that some do not agree with us, among others, Schwatt, who writes:

"The frequency of intestinal tuberculosis in the incipient and early stages of the pulmonary disease is difficult to determine. We believe that it is rather rare. Among the 2000 guests admitted to Bedford Sanatorium, the great majority of whom were early and moderately advanced favorable cases, clinical symptoms, on which a diagnosis of this complication could be ventured, were present in 16, of which 5 were moderately advanced, rapidly progressive, and 11 far advanced. Among the far-advanced patients in the wards of the Montefiore Hospital, about 25 per cent present definite symptoms of tuberculous enteritis." It is evident that Schwatt did not study these cases roentgenologically.

Many medical articles and even books have been written on the digestive disorders of patients with pulmonary tuberculosis. Our studies have convinced us that, many times, the cause of the frequent functional disorders of the stomach and intestine is to be found in the tuberculous ulceration in the intestines. Roeder, of the Hamburg Sanatorium, found them present in 67 per cent of the first,

21 per cent of the second and in 72 per cent of the third stage. The usual treatment of the digestive disorders in many of these patients leads often to no permanent benefit. If an early diagnosis of intestinal tuberculosis can be made and suitable treatment instituted at once, we feel sure that many such cases, now doomed to death, might make a good recovery. The marked improvement noticed in the lungs of some patients, who are recovering from intestinal tuberculosis, inclines us to lend support to the claim that intestinal ulceration is a frequent cause of reinfection of other parts of the body, especially the lungs, and hence an added danger of "entero-phthisis."

The pessimistic attitude of many physicians toward intestinal tuberculosis recalls the similar attitude expressed toward pulmonary tuberculosis twenty-five or thirty years ago. That the prognosis is very clouded no one can deny, but that many cases, when diagnosed early and submitted to appropriate treatment, do recover, can be easily proved by figures that we shall present (see p. 241).

Autopsy figures show that at death intestinal tuberculosis occurs in from 50 to 80 per cent, or even more, of all cases of pulmonary tuberculosis. This means for example that of 4780 patients who died from pulmonary tuberculosis in New York City during 1924, 2300 to 3800, or more, had intestinal tuberculosis, a complication seriously affecting their nutrition and, *a priori*, their chance of recovery. In Paris in 1922, 6884 persons died from pulmonary tuberculosis and, of these, 3400 to 5500 or more, had intestinal tuberculosis. Calculated in a similar manner, from 40,000 to 63,000 persons with pulmonary tuberculosis had intestinal tuberculosis at death in the United States in 1923, and from 16,000 to 25,000 in England and Wales in the same year.

Drolet has estimated that about 18,000,000 patients died from pulmonary tuberculosis throughout the world during forty years. Of this number, from 9,000,000 to 14,000,000 had intestinal tuberculosis.

Mention has been made of the greater danger of blood invasion in tuberculosis, when tuberculous ulceration occurs in the bowel, as the intestinal ulcers are probably the chief source of the blood infection in pulmonary tuberculosis. Fischer, Engelsmann, Senckenberg, J. Strauss, and Betke hold this view.

It is well known that it is difficult to infect some experimental animals by feeding and, furthermore, that it requires, some hold, 2000 times as large a dose as is needed for infection by inhalation. Tubercle bacilli can pass through the intestinal wall without producing changes in the wall and so gain entrance to the blood or

lymph stream. In pulmonary tuberculosis, tubercle bacilli if present in the sputum are practically always swallowed and B. Fischer thinks are often in part absorbed. He mentions the case of a woman, aged fifty years, with a tuberculous lung cavity, who always swallowed all her sputum. Tubercle bacilli were found in masses in her feces. At autopsy not a single intestinal ulcer was found but separate, equally distributed nodules, the size of hemp seed, were found everywhere in and under the mucosa. A subacute miliary tuberculosis, was the cause of death. In intestinal tuberculosis, Fischer thinks tubercle bacilli are always to be found in the thoracic duct even when it is normal. Without intestinal tuberculosis this does not occur. Attention is called to the small amount of lymph held in the thoracic duct and the large amount that passes through it, and, consequently, he states, that the body must be saturated with tubercle bacilli in ulcerative intestinal tuberculosis, when once the mesenteric glands have become tuberculous. The involvement of these glands necessarily slows the lymph flow in the intestines and so brings about extension there of the tuberculous process.

In 21 cases with tubercle bacilli in the thoracic duct studied by Betke, 20 had intestinal tuberculosis (4 miliary tuberculosis, 17 chronic pulmonary tuberculosis) and the other case had miliary tuberculosis with rupture of a tuberculous mass into a pulmonary vein. The 1 case without intestinal tuberculosis had also, possibly, intestinal tuberculosis but it was not proved. The mesenteric glands, in cases with tubercle bacilli in the duct, have all more or less severe tuberculosis. Of 9 cases without tubercle bacilli in the duct only 1 had miliary tuberculosis and 4 had no intestinal tuberculosis. While 3 of the cases had a fresh pleurisy and tubercle bacilli in the duct, in the others there was no question of tubercle bacilli coming from the pleura. Both Betke and Fischer believe that not only a primary, but also a new reinfection of the lung, producing a rather disseminated, rather finely nodular but not truly miliary type of the disease, can arise in this way. Intestinal tuberculosis can thus produce a vicious circle.

Tubercle bacilli are found at autopsy also in the portal vein. Fischer never found them there unless intestinal tuberculosis was present. Strauss found tubercle bacilli in the portal vein in 11 of 12 cases with intestinal tuberculosis and, in the negative case, the ulcers were scattered in the small intestine. He believes that in almost every case of intestinal tuberculosis, tubercle bacilli penetrate into the portal vein, usually in considerable numbers. He has not

found them in the absence of intestinal tuberculosis and, indeed, apart from miliary and generalized tuberculosis, has never found tubercle bacilli in the blood except with ulcerative intestinal tuberculosis. So he concludes that intestinal tuberculosis is the chief source of blood infection in chronic tuberculosis. Lancereaux holds practically the same opinion and Ullom confirms it from his work.

It is difficult to explain why the liver is not more often tuberculous under such conditions. Fischer believes that it is often affected, for miliary tuberculosis of the liver is common in pulmonary and intestinal tuberculosis, but does not tend to advance, and is, at times, easily overlooked. Weigert held that liver tubercles were found in advanced pulmonary tuberculosis as frequently as intestinal tuberculosis. Bristowe, in 1858, found tuberculous cavities of the liver in 17 of 167 cases of tuberculous ulcers of the intestines. Zehden, in 1879, found tuberculosis of the liver as frequent as ulcerative tuberculosis of the bowels. Rolleston thinks tuberculosis of the liver occurs in less than 50 per cent of ulcerative intestinal tuberculosis. Ullom, in 1909, in 200 livers of tuberculous patients, found tubercles 79 times and, in 45 cases with tubercles of the liver, ulcerative intestinal tuberculosis was present in 38.

Koizumi found tuberculosis of the liver in 11 of 12 cases, macroscopically normal. He studied the liver in 50 cases, in 23 of which, he had found tubercle bacilli in the feces and obtained the following results:

TABLE II.

		Liver normal macroscopically.	Miliary tuberculosis.	Steatosis.	Amyloidosis.
Tubercle bacilli in feces	+23	12	2	7	2
	−27	10	3	11	3
		22	5	18	5

In 29 cases with intestinal tuberculosis he found:

Tubercle bacilli in feces	+13	7	1	4	1
	−16	6	0	8	2
		13	1	12	3

Engelsmann found in 8 cases, dying of pulmonary tuberculosis without intestinal tuberculosis, only 1 with a tuberculous complication, nephritis, while, in 100 with intestinal tuberculosis, he found meningitis in 5, nodules in the spleen in 4, in the renal cortex in 6, in the epididymis, seminal vesicles and prostate in 4, and acute general miliary tuberculosis in 3.

Such observations emphasize the great importance of intestinal tuberculosis. The reinfection of the lung through the lymph and blood stream and the marked improvement of the pulmonary condition in some patients, who are subjected to proper treatment for their intestinal tuberculosis, indicates that a vicious circle can arise in intestinal tuberculosis. Furthermore, the constant infection of the lung and liver may influence adversely whatever immunizing processes exist. The danger, also, of miliary tuberculosis from absorption of tubercle bacilli from ulcerative intestinal tuberculosis is real.

Many surgeons will in the future refuse to operate for an abdominal condition, in patients with pulmonary tuberculosis, until intestinal tuberculosis has been excluded.

TABLE III.—INCIDENCE OF SECONDARY INTESTINAL TUBERCULOSIS.

Observers.	No. of cases of pulmonary tuberculosis.	No. of cases with intestinal tuberculosis, per cent.
Andral	....	80
Biedert	3104	40.7
Bollinger	....	70-80
Bayle	....	68.0
Coats	....	66.0
Conrath	....	85.0
Deist	....	75.0
Eichhorst	462	29.9
Eisenhardt	1000	567 (56.7)
Engelsmann	108	92.6
Fenwick and Dodwell	833	500 (57.0)
Fowler and Godlee	382	296 (77.4)
Frerichs	....	83.0
Harnan	832	68.0
Heinze	1226	51.0
Herxheimer	....	90.0
O. Kieffer	629	452 (72.0)
Lebert	....	67.0 in Breslau 39.0 in Zurich
Louis	125	82.0
Orth	....	70.0
Rousseff	800	46.0
Schwatt	199	130 (65.3)
Weigert	....	90.0
Walsh	100	76.0
Willigk	....	49.6

CHAPTER XII.

THE SITE OF THE LESIONS IN INTESTINAL TUBERCULOSIS.

Intestinal tuberculosis occurs most frequently in the ileo-cecal region, as many have pointed out. Why this should be so has already been fully discussed. This site is also, in a great majority of cases, the earliest seat of the disease. Some have even gone so far as to state that, if no tuberculosis exists in the ileo-cecal region, intestinal tuberculosis can be excluded, which of course does not always hold.

Fenwick and Dodwell, in a study of the sites of the ulcerative lesions in 500 necropsies, found the ileo-cecal region affected in 85 per cent (confined to this region in about 10 per cent), the jejunum in 28 per cent, the duodenum in 3.4 per cent, the ascending colon in 51.4 per cent, the descending colon in 21 per cent, the sigmoid in 13 per cent and the rectum in 14 per cent. Fowler and Godlee, in 323 cases, found the following parts affected with ulceration: Duodenum in 2.1 per cent, jejunum in 21 per cent, ileum in 59 per cent, cecum in 59 per cent, appendix in 41.7 per cent, colon in 41.8 per cent, rectum in 13.6 per cent. In 382 cases, they state the small bowel was affected alone in 8.6 per cent cases, the large bowel alone in 16.4 per cent and both in 52.4 per cent. Though the appendix is involved in about 70 per cent in some statistics, acute appendicitis rarely occurs.

Powell and Hartley, in a study of 160 cases, found the duodenum affected in 3.8 per cent, the jejunum in 20.3 per cent, the ileum in 47.8 per cent, the cecum in 43.6 per cent, the appendix in 37.5 per cent, the colon in 31.8 per cent, the rectum in 13.4 per cent. In 7.2 per cent it was found only in the small bowel, in 9.2 per cent only in the large bowel and in 41.7 per cent it occurred in both large and small bowel.

Engelsmann found in his 100 cases that the small bowel was involved in 91, the large in 83, both in 75 and the appendix in 70. The whole bowel was affected in 57, the large and small bowel in 18, the small bowel and appendix in 8, the large bowel and appendix in 4. When the disease was isolated he found it 8 times in the small,

4 times in the large bowel and once in the appendix. It occurred in the ileum alone in 34, in the ileum and jejunum together in 51 (ileum greater than jejunum 16; ileum less than jejunum 9), and in the jejunum alone in 6. The duodenum was affected 9 times.

Frerichs found the ileum affected alone in 35 per cent, the colon alone in 3 per cent, both in 45 per cent, and the ileo-cecal region involved in 80 per cent.

Lebert found the small bowel diseased alone in 42 per cent, the large in 7 per cent.

Walsh in 76 cases found:

TABLE IV.

				Showing no symptoms.
Jejunum alone	2			1
Colon alone	5	=13		3
Small gut	6		76	1
Cecum alone	6			2
Large	8	=14		2
Both		49		17

Schwatt, in collaboration with Steinbach, in an excellent and careful study of 199 cases of pulmonary tuberculosis at the Montefiore Hospital, reported 130 cases with ulceration and 4 cases with tubercles and no ulceration. The small intestine was involved 109 times (82.5 per cent) and alone in 30 (23.4 per cent). The large intestine was involved 98 times (76.2 per cent) and alone in 19 (15 per cent). Both were ulcerated in 80 (62.5 per cent). In 125 cases ulceration occurred in the duodenum in 3 (2.4 per cent), in the jejunum in 43 (34.4 per cent), in the ileum in 105 (84 per cent), in the cecum in 84 (67.27 per cent), in the appendix in 40 (32 per cent), in the colon in 63 (50.4 per cent), and in the rectum in 15 (12 per cent). The ileum was the segment most frequently involved alone and most frequently ulcerated. In 2 cases, there were tubercles only without ulcers. In over 75 per cent, there were only one or two small superficial ulcers and in only 2 cases was extensive involvement with advanced ulceration present. In 80 cases with disease of large and small bowel, the cecum was involved in 71. In 5 cases, it was alone diseased. In 2 cases, there were tubercles only. In 63 cases of ulceration of the colon, the ascending colon was involved in 50, the transverse alone in 2, and this and other distal portions in 4 cases. In 8 cases, the cecum was not diseased and in the majority of cases, the disease in the colon was apparently an extension of the disease from above. In fact Westenhoefer, he says, believes that if the ileo-cecal region is not tuberculous, intestinal

tuberculosis can be excluded. Progression of the disease to the large bowel (80 out of 86 cases) appears to occur earlier than to the small intestine (44 out of 86 cases), due, Schwatt suggests, to the fact that the tubercle bacilli coming from the ulcers in the bowel are more virulent than those passing through the stomach, which is, of course, far from being a proved fact.

The tendency of intestinal tuberculosis to limit itself to the ileo-cecal region has led Benoit to divide intestinal tuberculosis into two groups: (1) Where it is limited to the ileo-cecal region, and, (2) where the disease is widely disseminated through much of the bowel. It appears to us, however, that one is simply the later stage of the other, and depends, among other things, upon how long before death the intestinal tuberculosis developed, how rapidly the pulmonary lesion advanced after the bowel was involved, the time of occurrence of fatal complications, etc. In tuberculosis in man, the involvement with tuberculosis of the ileo-cecal region ranks next in frequency to that of the lungs. Lesions in the colon nearly always accompany those in the small intestine but may occur alone (MacCallum).

It must not be overlooked that all these figures are based only upon autopsy records.

CHAPTER XIII.

PRIMARY AND SECONDARY INTESTINAL TUBERCULOSIS.

Primary intestinal tuberculosis has proved to be very rare. Since the separation of the bovine and human types of tubercle bacilli, much work has been done and figures collected to show the prevalence of primary intestinal tuberculosis. It is supposed to be more common in children, and Heller found it 10 times in 85 autopsies in children and, again, 6 times in 28 similar cases. Bonome, in 4224 autopsies (performed between 1889 and 1906), found 126 cases of primary intestinal tuberculosis in 769 cases of tuberculosis. Biedert found it 16 times in 3104 cases of tuberculosis and, at the Munich Pathological Institute, it occurred once in 1000 cases. Bovaird, in 1481 collected cases, found 150 with primary intestinal tuberculosis. He calls attention to the fact the English have many, the Americans few cases of primary intestinal tuberculosis.

In a series of 22,725 autopsies, collected by Gant, and including many of the foregoing figures, primary intestinal tuberculosis was said to be present in 7.22 per cent. One case occurred in our experience with adults. Mummery has never seen a case of primary tuberculosis of the colon. In 100 cases of hyperplastic tuberculosis of the colon, the disease was primary in 76, secondary to pulmonary tuberculosis in 18 per cent.

While the method of diagnosis to be described is applicable also to these cases all that can be said safely, without further data, is that an ulcerative condition is present.

It is of interest to note that Weber and Tante found bovine tubercle bacilli in 25 of 28 cases of primary intestinal tuberculosis.

CHAPTER XIV.

CLINICAL SYMPTOMS OF INTESTINAL TUBERCULOSIS.

The clinical symptoms of intestinal tuberculosis are extraordinarily variable and, unfortunately for both patient and physician, resemble, at the outset, very closely those produced by fleeting disturbances of no moment. In fact, no abdominal symptoms, whatsoever, may be noted even though extensive disease may be present throughout both large and small bowel. It is striking that such a condition can exist without discovery, even when acute observers are on the lookout for it. It must not be forgotten that, while secondary intestinal tuberculosis may occur at any time in the course of pulmonary tuberculosis, it occurs usually in the more advanced stages of the disease, when it is often difficult to decide which symptoms are due to the ulcerative entero-colitis and which to the pulmonitis. The intestinal symptoms can dominate the picture as in a patient of ours, who was referred to us by a surgeon to whom in turn he had been referred. Gaither notes that only gastro-intestinal symptoms may be present in pulmonary tuberculosis, but fails to state whether or not intestinal tuberculosis was excluded. Cases have occasionally been confused with typhoid fever, which, some believe, occurs when secondary infection of the tuberculous ulcers with other organisms, *e. g.*, staphylococci, takes place.

In any case then, slight digestive or abdominal symptoms should always suggest the possibility of intestinal tuberculosis and, if they persist, a more careful study should be made to exclude intestinal tuberculosis. When any patient, even in the incipient stage, begins to do poorly and shows no increase of pulmonary symptoms or signs or, what is even more significant, presents a lessening of signs and symptoms referable to the lungs, intestinal complications should be borne in mind. Further, if under these circumstances the patient exhibits extreme nervousness, constipation, slight dyspepsia, a feeling of fullness or discomfort in the abdomen after eating, accompanied in all probability by gas, so frequent in all patients undergoing treatment for tuberculosis, or fails to regain lost weight under suitable conditions, the probability of intestinal

complications is strengthened. These symptoms may be accompanied, at times, by a very slight and brief attack of diarrhea, readily amenable to simple remedies. A slight irregular rise of temperature may or may not be present.

In some instances the patient complains that he does not feel well; his digestion is neither good nor bad; and he may have a slightly irregular temperature, often normal, but at times reaching 99.6° F. in the afternoon. In one such case, a slight thickening of the cecum and ascending colon was palpated, and at operation tuberculosis of the cecum and ascending colon was found, and the bowel resected with good results.

In another case, with incipient pulmonary lesions, marked constipation was accompanied, at times, by persistent vomiting or regurgitation of food. A palpable cecum suggested intestinal tuberculosis, which was confirmed roentgenologically and at operation, with recovery after excision.

As the disease progresses, the symptoms usually increase and depend on the location of the ulcers, the irritation of the nerves, the involvement of the peritoneum, the condition of the mucous membrane and, of course, on the changes in the intestinal contents produced by the ulceration (blood and pus).

The point that we would like to emphasize is that, at the onset, the symptoms of intestinal tuberculosis may be as slight and as indefinite as those of early pulmonary tuberculosis. In fact, the term "latent" might well be applied to the majority of cases of intestinal tuberculosis, for, unless borne in mind and looked for, they escape detection for a longer or shorter period.

PAIN IN INTESTINAL TUBERCULOSIS.

Pain and diarrhea are the two most common symptoms of intestinal tuberculosis.

Pain is by no means present in all cases of intestinal tuberculosis, but is more characteristic than that due to other forms of ulceration. It may even be absent, or so slightly marked, as to be overlooked in cases with the most pronounced ulceration. In many cases where it does occur, it is not an early symptom. It may be preceded by slight discomfort, noticed more particularly after meals. A little later, some pain, just at or below the epigastrium, about the umbilicus, or most frequently in the right lower quadrant, may occur at irregular intervals, often later in the morning or in the afternoon, and is at first transient. The pain gradually becomes more per-

sistent, recurring from day to day, and resembling that of neuralgia, may encircle the body or may be crampy or stabbing in character (suggesting gas), aggravated by food, and relieved at times by fasting. Sharp and, later, lancinating pains may follow a glass of cold water or milk, due to increased peristalsis. In some instances the pain is aggravated by pressure (palpation) or, indeed, only elicited by such a procedure or by rapid decompression after pressure. The ileo-cecal region is nearly always sensitive to pressure. Pain, of course, may, but does not usually precede the diarrhea. As the disease progresses, the attacks become more frequent and may occur several times in the day.

It is possible, as Schwatt indicates, that all latent cases would develop pain, diarrhea or other symptoms, were it not for weakness or the occurrence of death, due to the pulmonary disease.

Pain is due, many think, to involvement of the peritoneum (serositis) and may, in such cases, be severe or localized, and combined with tenderness, nausea and vomiting, and rise of temperature. The pain may be elicited only on pressure and, here, some observations would suggest that such pain bespeaks an involvement of the appendix as well. In severe pain the legs are often flexed to relieve the pressure, due at times to local or general distention.

Pain is usually brought about by abnormal motor activity in connection with some resistance to the onward movement of the intestinal contents (spasm, stenosis) or to localized peritonitis. Pain, immediately after or at the time of eating, is due usually to reflex peristalsis in the ileum or colon. It may also be due to non-tuberculous enterocolitis or to abnormal fermentation.

Involvement of the mesenteric glands, occurring in about 50 per cent of the cases, is at times accompanied by excruciating pain, which may be described as throbbing and burning. The tenderness may be localized, a mass felt, but at other times nothing definite can be made out. Calcified glands may also cause pain.

Engelsmann noted pain in 22 of his 100 cases, 19 with general ulceration and 3 with ileo-cecal disease. It was stated to be abdominal in 13, in the stomach in 4, in the right lower quadrant in 4 and under the ribs in 1. Peritoneal adhesions apparently caused the pain in 3 and tuberculous peritonitis in 2.

THE BOWEL MOVEMENTS IN INTESTINAL TUBERCULOSIS.

The association of diarrhea with intestinal tuberculosis is firmly fixed in the minds of most medical observers. Such, however, is far

from being the case for in no small percentage diarrhea never occurs and constipation or even obstipation may prove most troublesome.

Diarrhea.—In some instances, the first manifestation of intestinal tuberculosis may be the disappearance of a previous chronic constipation. The patient remarks, often with much joy, that his "bowels are better" because they move now without drugs. A little later the stools may become mushy and occur once or twice at night. The diarrhea, which next sets in, may be so transient and so easily remediable as to attract no attention. But the occasional attacks before long increase in number, become more severe, and the diarrhea becomes continuous and persistent. Some patients may never have more than one loose stool a day. It must never be forgotten that patients with pulmonary tuberculosis can have attacks of diarrhea lasting for six or even eight weeks, due to other causes than intestinal tuberculosis. Chronic, non-tuberculous diarrhea can of course occur as in one of our patients who had no ulcerative colitis.

Lebert was of the opinion that diarrhea without ulceration occurred in from one-tenth to one-seventh of all cases coming to necropsy and Louis found ulceration in only 50 per cent of his patients who had had diarrhea. He felt that in a patient with pulmonary tuberculosis, the diarrhea must be chronic, continuous and frequent, in order to enable one to say definitely that ulceration was present, which is further upheld if the stools are foul.

It is not unusual at the outset to have the diarrhea alternate with constipation. In the later stage the diarrhea may be very profuse, the stools frequent and large, and, apparently by "dehydration," reduce the amount of the expectoration and the number of night-sweats, which, some think, may alternate with the diarrhea. This is the colliquative diarrhea of Bayle, the intestinal "sweats" of Graves. The number of stools daily in some cases may reach ten or even more.

Graves reports a case with extensive ulceration with constipation and without symptoms. "Our predecessors entertained" he goes on to say "a notion that the diarrhea of phthisis is a species of internal sweating; they observed that when the patient ceased perspiring from the skin, he was generally attacked with a watery diarrhea and hence they termed the diarrhea, colliquative. Afterward, it was found, on numerous examinations, that where this diarrhea had existed, there was, in most cases, ulceration of the bowels; hence pathologists began to believe that this ulceration had a great deal to do with the intestinal symptoms observed toward the termination of phthisis, referring to it the abdominal pain and

tenderness, the unmanageable character of the diarrhea, and the aggravation of the hectic symptoms." Graves thinks this idea was possibly too hastily adopted.

The cause of diarrhea is either increased peristalsis or interference with the absorption of water from the intestinal contents. Among other things producing increased peristalsis may be mentioned the irritation of swallowed sputum, the chilling of the abdomen, especially at night, nervousness, raw food, especially fruit, food that produces gas, and overfeeding; but usually these causes are concerned more often with the remittent type of diarrhea. Nothnagel holds that diarrhea is associated with faulty absorption of water in the more distal parts of the colon. The intestinal contents in health remain in contact with the mucous membrane of the large bowel from 5 to 29 hours and ample opportunity is thus given for absorption of water.

It is still an open question how much importance should be attributed to the ulceration, how much to the inflammatory changes (see p. 62), to the associated catarrhal condition, and how much to the amyloid degeneration present at times. The relation of the site of the lesions to the diarrhea is difficult to state.

Schwatt believes that ulceration of the small intestine, cecum and ascending colon never produces diarrhea unless associated with one of the changes just mentioned. Ulceration of the lower segments of the colon and of the rectum interferes with the absorption of water and invariably causes diarrhea, but, almost immediately afterward, he says that diarrhea may be absent in severe and extensive ulceration of the colon because the irritability of the nerves having become inhibited or abolished by such marked destruction of the bowel wall, no increased peristalsis occurs.

All who have studied intestinal tuberculosis have come to the same conclusion, namely, that the number, size and depth of the ulcers play often a part secondary to other conditions such as catarrhal or inflammatory changes, produced often by the causes mentioned, viz., swallowed sputum, indiscretions in diet, etc.

Constipation.—In the minds of most physicians constipation is seldom linked with intestinal tuberculosis but it is by no means infrequent. In some cases, constipation does not exist for the bowel is entirely empty at the end of twenty-four hours and still the patient feels "constipated." In one such case, where a definite ulcerative condition existed in the hepatic flexure, the patient was always anxious to take strong purgatives, which apparently relieved this feeling, though roentgenological study showed the bowel to be

empty. The spasm, which may cause definite constipation, is no doubt the cause of this feeling and also of the stubborn obstipation that some describe. Dyschesia, of course, may readily occur late in the disease.

Engelsmann found constipation in 18 of his 100 cases, 7 times with general ulceration, and 8 times with ileo-cecal disease, twice with lesions in the small bowel and once in the large. He thinks it a valuable sign of ileo-cecal tuberculosis. Constipation was noted by Girode, Hanot, and many others.

In a study of 300 cases of pulmonary tuberculosis, Harris and Beale found 132 (44 per cent) had some bowel irregularity (in 100 early cases it occurred 27 times; in 100 more advanced 46 times; and in 100 late cases 59 times).

THE TEMPERATURE IN INTESTINAL TUBERCULOSIS.

The temperature may be normal or elevated, but is often subnormal at some time of the day, usually in the morning. It frequently runs no regular course, the curve varying irregularly from day to day. Again, the temperature may be normal for some days and then reach 99.4° or 100° F. for a day or possibly two. Such a course is very unusual in uncomplicated pulmonary tuberculosis. It has been stated that withholding food for a day or more may reduce the fever if due to the intestinal ulceration. On the other hand, the temperature does rise at times without any increase of pulmonary symptoms or signs, which is always suggestive.

The pulse-rate is increased in most cases but it is difficult to associate the hurrying with any special influence of the intestinal tuberculosis other than that which causes rise of temperature.

GASTRIC SYMPTOMS OF INTESTINAL TUBERCULOSIS.

Gastric symptoms may be absent or predominate the clinical picture. In Walsh's 100 cases he found:

TABLE V.

	Ulceration of				
	Small bowel.	Large bowel.	Both bowels.	Ulceration, per cent.	No ulceration, per cent.
Gastric disturbance	2	5	15	22 (29.0)	5 (21.0)
No gastric disturbance	11	9	34	54 (71.0)	19 (79.0)

Pritchard, in a study of the stomach contents of 15 cases by the fractional method, found the acidity within normal limits in 12,

low in 1, and in 2 no free hydrochloric acid and a low total acidity. Erickson noted distress after eating, sour stomach and acid eructations, as the most frequent troubles. Lemon noted fermentation (gas) and the symptoms mentioned by Erickson. Anorexia, gas and discomfort after meals were each present in half or more of Logan's cases.

In the 100 cases that Erickson studied, nausea was present in 52 and vomiting occurred in 37. Among the other digestive disturbances, he found distress after eating (28), disturbances of appetite (26), sour stomach (20), acid eructation, water brash, etc. (14) and heartburn (3).

Occasionally, the only symptom may be persistent vomiting, even of liquids. One such case had a definite cecal tuberculosis at operation, which was followed by recovery, and a second, at autopsy, showed extensive ulceration throughout both large and small bowels. Some pathological changes are said to occur in the stomach in nearly all cases of pulmonary tuberculosis complicated with intestinal tuberculosis (see p. 66).

LOSS OF WEIGHT IN INTESTINAL TUBERCULOSIS.

In no phase of the disease does emaciation progress more rapidly than when anorexia and weakness supervene upon severe, persistent diarrhea with many stools (colliquative diarrhea). The body seems to melt away and several pounds a day may be lost for some days.

A study of the weights in Lemon's cases showed that 11 patients with constipation had lost 15 per cent, 9 with diarrhea had lost 18 per cent, 3 with alternating constipation and diarrhea had lost 17 per cent, 12 patients with apparently normal bowels had lost 15 per cent. Two patients with a history of tarry stools had lost 30 per cent. He says he could note no great variation in loss of weight, regardless of the condition of the bowels.

INTESTINAL HEMORRHAGE IN INTESTINAL TUBERCULOSIS.

Intestinal hemorrhage is of rare occurrence in intestinal tuberculosis, but macroscopic blood is not uncommon (13 per cent, Erickson). Fatal cases have been reported. J. C. Cruice has collected 26 cases of large hemorrhages and added 4 fatal cases. Engelsmann reported 2 fatal cases, 1 arising in the small bowel,

1 in the large. Death usually follows in a few days after a recurrence of the hemorrhage, as occurred in 1 of our cases. It is possible that the slow development of the ulceration may give ample time, in most cases, for the obliterative endarteritis or thrombosis to close the vessels before their walls are destroyed (see Complications, p. 217).

Some patients have marked mental depression and great and often unaccountable nervousness. Acute intestinal tuberculosis has been described with symptoms suggestive of acute endocarditis or sepsis. We have not encountered any such cases, and the acute cases, seen by us, are usually due to a tuberculous appendix. Intestinal tuberculosis is rare in acute tuberculosis. When the pulmonary symptoms are masked by the intestinal symptoms, the condition can be confused with typhoid fever, which is well illustrated in the following case.

"Dr. Simpson showed the lungs and a portion of the intestine from a case of tuberculosis. The interest lay chiefly in the close resemblance to enteric or typhoid fever. Mary P., aged twenty-one years, domestic servant, was admitted to the Manchester Infirmary, November 17, 1881. The health had been good up to six months before. Since then she had felt increasing weakness, but became much worse three or four weeks before admission, with cough and expectoration. On admission, the evening temperature was 104° F., pulse 128. There were morning remissions. Slight flattening, with diminution of resonance and some moist rales, existed at the left apex. Sonorous and cooing rhonchi and moist rales were heard, indeed, over the greater part of the chest, but the latter were constant at the left apex. The abdomen was natural, not tumid. There was no pain nor gurgling on pressure in the iliac fossa; but a few rose spots, exactly like those of typhoid, were scattered over the surface. The tongue was a little furred, but moist. The bowels were slightly confined. The spots appeared and disappeared in successive crops through the whole course of the case. There was high temperature in the evenings with morning remissions. On November 27, diarrhea set in and the motions speedily assumed the 'pea-soup' character. The expression, the mental hebetude, the character of the delirium all closely resembled what is seen in typhoid. She died on December 18. Both lungs were congested at the bases. A small cavity, the size of a walnut, was found at the left apex, while numerous miliary nodules were scattered through the surrounding lung-tissue. In the middle lobe

of the right lung was a caseous mass of the size of a filbert with some scattered miliary tubercles. The peritoneum was normal, and also the mucous membrane of the ileum till near the lower end, where numerous small rounded and oval ulcers were found. Their margins were thickened; the floor was formed by the muscular, and in some cases the serous coat, but there was no perforation. The ulcers extended transversely around the intestine, contrasting markedly with those found in the typhoid. They extended through the whole length of the large intestine. The other organs were healthy."

From a study of 130 cases, Krag-Möller, in 1921, found a causal connection between tuberculosis of the small intestine and anemia, due, he thought, to absorption of hemolytic poison toxins, produced by the tuberculous process in the small bowel. This is not true in the large bowel as there, he thinks, the diarrhea prevents absorption. Several cases later shown to be intestinal tuberculosis have presented the blood picture of pernicious anemia. These conditions, however, are very unusual, and none occurred in the cases we are discussing.

In his study of 45 selected cases of pulmonary tuberculosis and intestinal tuberculosis, J. E. Pritchard, in 1924, found pulmonary symptoms present in all. They had preceded the intestinal symptom in 39. In 5 the general breakdown was coincident with the intestinal symptoms. One had no intestinal symptoms. Nervousness was an outstanding symptom in 5 and fairly marked in 19 (42.4 per cent). Anorexia, complained of by 32, was the first symptom in 15. Some stated that the appetite was good but a few mouthfuls satisfied it. Discomfort was present in 33 (73.3 per cent) and was the first local symptom in 7. It was commonly associated with anorexia and nausea. Definite pain was present in 42, and was the first symptom in 5. It was rarely absent in advanced cases, was usually crampy, confined in two-thirds to the lower abdomen, and often attributed to gas. The pain was frequently so slight as to cause no complaint, but often persisted from day to day. Flatulence, present in 26 cases, was not an early symptom. Nausea occurred in 19 and vomiting in 10. Constipation was complained of by 19 and was the first symptom in 13. One case had marked constipation, followed by diarrhea, and at operation had ulceration only of the large bowel. Diarrhea ranked next to pain in frequency (40) and usually came on insidiously. It alternated with constipation in some, was associated with pain in all but 2 cases and was always a marked symptom in advanced cases.

Lemon reports a study of 36 operative cases of A. H. Logan's which are given in the following table:

Findings in 36 cases of tuberculous enterocolitis reported by Logan:

TABLE VI.—(LOGAN'S STUDIES).

Age, years.	Per cent.	Sex.	Cases.
20 to 30	30.5		
30 to 40	33.3	Males	17
40 to 60	30.5	Females	19

Duration of symptoms.	Per cent.
Less than 1 month	52.0
1 to 3 "	28.9
4 to 6 "	11.1
10 "	1.0
12 "	1.0
20 "	1.0

Symptoms.	Per cent.
Alternating constipation and diarrhea	11.1
Diarrhea	16.6
Nausea	72.2
Constipation	50.0
Vomiting	64.0
Onset with acute attack of abdominal pain	50.0
Onset with a history resembling appendicitis	50.0
Pain:	
Present	89.0
Absent, 1 case; not noted, 3 cases	0.00
Character of pain:	
Soreness and tenderness	18.0
Crampy	64.0
Not noted in 8 cases	
Discomfort and symptoms after meals	49.0
Nervousness in 5 cases.	
Gas	58.0
Anorexia	58.0

Previously operated on elsewhere, 21 cases; no improvement in 20.

Baker in a study of 95 cases of which 91 had symptoms, found by roentgen-ray study 21 (22 per cent) were positive, 17 (18 per cent) suspected, and 57 (60 per cent) were negative. The symptoms were as follows:

TABLE VII.

	Intestinal tuberculosis.	Suspected.	Negative.
Diarrhea	15	7	6
Abdominal pain	18	7	12
Gas	7	8	26
Constipation	1	3	11
Anorexia	3	7	16
No complaint	1	1	6

CHAPTER XV.

CLINICAL SYMPTOMS OF INTESTINAL TUBERCULOSIS (*Continued*). ERICKSON'S WORK.

In this chapter are to be found the results of a study of 100 cases, proved by roentgen-ray to have intestinal tuberculosis and of 40 cases with intestinal symptoms, shown by roentgen-ray to be free from intestinal tuberculosis. The study was undertaken at our suggestion by R. J. Erickson and all the cases had been studied in the roentgen-ray laboratory at the Trudeau Sanatorium. We feel that this careful, detailed investigation adds information obtainable nowhere else and those, interested in the subject, should devote some time to this admirable piece of clinical investigation.

DURATION.

Erickson found that, in the majority of positive cases (68), the pulmonary tuberculosis had existed one or more years, and in 36 cases over two years, before symptoms of intestinal tuberculosis had occurred. In 10 cases the symptoms had occurred simultaneously and in 2 the intestinal symptoms preceded the pulmonary. Thus, based on the occurrence of symptoms, the intestinal disease

Preceded the pulmonary disease in	2
Occurred simultaneously in	10
Occurred during 1st year in	20
Occurred during 2d year in	29
Occurred during 3rd year in	22
Occurred during 4th year in	9
Occurred after 4th year in	4

In other words symptoms of intestinal tuberculosis developed in 30 during the first year of the pulmonary tuberculosis, in 59 per cent within two years and in 81 per cent within three years.

PREVIOUS GASTRO-INTESTINAL HISTORY.

The previous gastro-intestinal history of these cases is of interest. Fifty-eight gave a negative history, 23 suffered from old chronic constipation (4 of these had a marked increase of constipation as a prominent initial symptom of their intestinal tuberculosis), 18 gave a history of varied general digestive disturbances (14 of these showed a marked increase at the onset of their intestinal tuberculosis, while

4 had no change), 5 gave a history of preceding attacks of diarrhea (in 3 this was a prominent symptom of their intestinal tuberculosis), 2 gave a history of and had as initial symptoms of their intestinal tuberculosis, nausea and vomiting. The foregoing patients had symptoms for several years, longer than it was possible to attribute to the intestinal tuberculosis, indeed, often going back to childhood. Three patients had had diarrhea off and on all their lives, and 1 patient had had nausea with her menstruation for years. It would appear from this that previous gastro-intestinal symptoms have a tendency to determine the initial symptoms of the intestinal tuberculosis.

It is of interest to note that the 17 cases, which had had chronic constipation and had no marked increase at the onset of intestinal tuberculosis, had as predominating symptoms at onset:

	Times.
Diarrhea	7
Pain	4
Nausea and vomiting	4
General digestive disturbance	2
Unexplained temperature	2
Violent combination of many symptoms	4
Nervousness	Once

It is difficult, basing the estimate on these figures, to attribute much importance to constipation, as it was also present in 28 per cent of the patients thought to have but really without intestinal tuberculosis. As the figures are small, too much significance on this point must not be attached to them. A similar statement holds true in regard to general digestive disturbances.

ONSET OF INTESTINAL TUBERCULOSIS.

The onset of intestinal tuberculosis was classified as insidious in 34 per cent of the cases, *i. e.*, the symptoms were so slight that it was difficult to determine a definite date of onset, or to say, whether or not, the symptoms were due to the pulmonary or intestinal tuberculosis, or, indeed, only an aggravation of long existing symptoms. In these 34 cases the insidious onset was characterized by minor digestive disturbances (12), minor constipation (8), slight looseness of stools (6), slight pain (3), slight fever (2), slight nausea (2), and considerable increase of nervousness (1). Unless borne in mind these changes would have been overlooked.

In 66 per cent of the cases, the onset was with quite definite symptoms; in 25 instances, where the symptoms were clear-cut, the onset was predominantly with pain, in 16 with diarrhea, in 8

with nausea and vomiting, in 5 with constipation, in 5 with fever and general constitutional disturbances, in 3 with general digestive disturbances, and in 4 with a severe or violent combination of many symptoms. Of course it must be remembered that the onset was named for the most prominent symptom. In no case, for example, was pain unaccompanied by other symptoms.

SYMPTOMS OF INTESTINAL TUBERCULOSIS.

In order to see if there was any predominating sequence, the symptoms were divided by Erickson, according to the time of their appearance, into primary, secondary and tertiary, which, however, has nothing to do with their intensity or relative prominence.

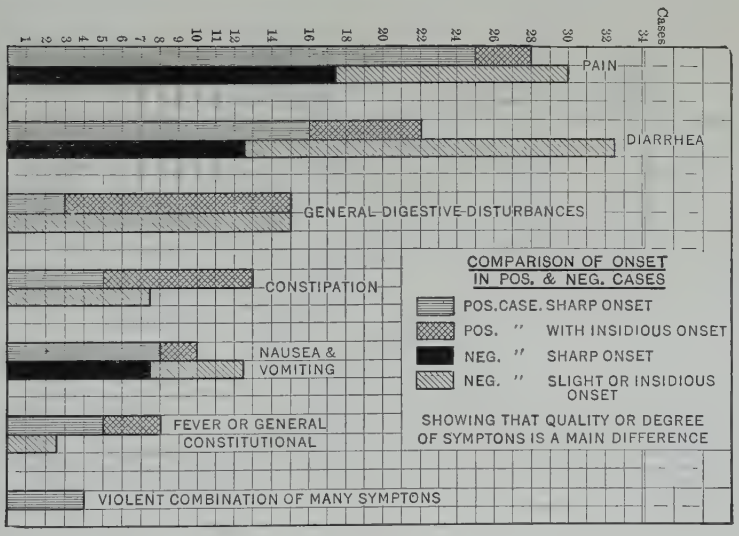
Cases
1 2 3 4 5 6 7 8 9 10 11 12 14 16 18 20 22 24 26 28 30 32 34
PAIN
DIARRHEA
GENERAL DIGESTIVE DISTURBANCES
CONSTIPATION
NAUSEA & VOMITING
FEVER OR GENERAL CONSTITUTIONAL
VIOLENT COMBINATION OF MANY SYMPTONS
COMPARISON OF ONSET IN POS. & NEG. CASES
POS. CASE. SHARP ONSET
POS. " WITH INSIDIOUS ONSET
NEG. " SHARP ONSET
NEG. " SLIGHT OR INSIDIOUS ONSET
SHOWING THAT QUALITY OR DEGREE OF SYMPTONS IS A MAIN DIFFERENCE

FIG. 16.—Comparison of symptoms in positive and negative cases.

Pain.—Pain was very carefully studied in regard to the period and time of its occurrence, its degree, character, location and whether or not accompanied by flatulence. It was found to occur in over one-third as a primary symptom (40 per cent), in a fourth as a secondary symptom, and only 7 had no pain until late in the disease. It was severe in 50, moderate or slight in 21, and therefore present in 71 per cent. It was crampy or colicky in 49, but soreness, deep aching, vague or sharp stabbing pains occurred with about equal frequency in the remainder. It occurred most often (31) in the

right lower quadrant, but was diffuse in 21, across the upper abdomen in 10, across the lower abdomen in 7, in the middle of the abdomen in 7 and in the left lower quadrant in only 2. It occurred after meals in 29, at night in 10, and at no special time in 24. In 39 cases with pain as an initial symptom, the following symptoms were present at the same time:

Diarrhea	17
General digestive disturbance	13
Nausea	14
Constipation	4
General systemic symptoms	3
Alternating constipation and diarrhea	1

A study of the duration of the pain showed that of the 71 cases, which had pain as a symptom, including 16 still having at the time of investigation pain, which had been present for varying lengths of time up to two years, 8 cases had suffered for over two years and 1 of them for five years. Seventeen had suffered pain for over one year; over two-thirds had had it less than six months.

The figures on which these statements are based are as follows:

TABLE VIII.—INCIDENCE OF PAIN.

	Pain present.	With flatulence.	Degree of pain. Severe.	Moderate or slight.
Primary period	39	30	28	12
Secondary period	25	18	16	6
Tertiary period	7	7	6	1
Total	71	55	50	19

Character of Pain.

	Cramping or colicky	Soreness.	Deep aching.	Vague.	Sharp stabbing.
Primary period	27	5	3	3	2
Secondary period	20	1	3	1	1
Tertiary period	2	0	0	1	2
Total	49	6	6	5	5

Location of Pain.

	Lower right.	Diffuse.	Across upper abdomen	Across lower abdomen	Middle.	Lower left.
Primary period	16	11	6	5	3	1
Secondary period	13	9	4	2	3	0
Tertiary period	2	1	0	0	1	1
Total	31	21	10	7	7	2

Time of Occurrence.

	After meals.	Before meals.	In A.M.	Night.	2 to 4 P.M.	During night.	No. special time.
Primary period	13	1	3	10	1	0	11
Secondary period	13	0	1	0	0	1	8
Tertiary period	3	0	0	0	0	0	5
Total	29	1	4	10	1	1	24

Diarrhea.—Diarrhea occurred in the primary period in 36, in the secondary in 19, and in the tertiary in 11 cases, totaling 66, thus being almost as frequent as pain. In 6, it was the only initial symptom, and lasted two to seven months before other symptoms developed. The looseness was most likely to occur in the morning in 22, at any time in 21, at night in 10, after eating in 6 and at 2 P.M. in 1 case. The number of stools was 1 or 2 in 8 cases, 3 or 4 in 30 cases, 5 or 6 in 13, 7 to 10 in 8, and more than 10 in 5 cases. When it occurred as an initial symptom it was accompanied by the following symptoms: pain, 17 times; general digestive disturbances, 13 times; nausea, 8 times; vomiting, 8 times; general systemic disturbances, 8 times. Diarrhea usually persists longer than pain.

Blood in the stools is more frequent than some suppose, occurring in 13 per cent. Definite bowel hemorrhage occured in 6 cases and was severe in 1.

Constipation.—Constipation was present in 28 cases, and in 9 as the sole initial symptom, where it had existed from one to seven months, and was still present in 2. It was followed by diarrhea 12 times, by nausea and vomiting 3 times, and by general digestive disturbances twice. It was accompanied by general digestive disturbances 8 times, general systemic disturbances (fever) 5 times, nausea and vomiting 4 times, and by pain 3 times. The figures on which these statements are based:

TABLE IX.

	No. of times present.	Character	
		Severe.	Moderate or slight.
Primary period	19	8	11
Secondary period	4	1	3
Tertiary period	5	0	5
Total	28	9	19

Duration of Constipation.

No. of months.	No. of cases.	Cases in which it still exists.
1	5	2
2	3	2
3	2	2
4	3	2
5	1	
6	1	
7	5	
8	1	
9	1	
14	2	1
18	2	1
3 years	1	1
4	1	1
5	1	1

It thus appears that constipation usually existed longer than pain, diarrhea or nausea.

Alternation of Diarrhea and Constipation.—The alternation of diarrhea and constipation, long known to exist in intestinal tuberculosis, was present in 12 cases. The relative importance of the two symptoms varied as can be seen from the following analysis of the cases:

One case of initial alternating diarrhea and constipation.

Duration.—Persistent for two years (one period of real diarrhea for a few days only).

Roentgen-ray.—Four series, March 1922 to August 1923. Ulceration involving cecum and ascending colon; small bowel suspicious; definite clearing.

Three cases of alternating diarrhea and constipation followed by diarrhea.

1. *Duration.*—Diarrhea and constipation six months.

Roentgen-ray.—December 1922. Involvement of cecum; May 1923, less evidence of involvement.

2. *Duration.*—Old history of constipation with spells of diarrhea since childhood; diarrhea for last four months.

Roentgen-ray.—November 1923. Involvement to hepatic flexure and questionably points distal.

3. *Duration.*—Loose stools with moderate constipation for two years; diarrhea for last four months.

Roentgen-ray.—May 1923. Probable ulceration of cecum and part of ascending colon; August 1923, irregular cecum, no spasm.

One case of initial alternating diarrhea and constipation, and followed by constipation.

Duration.—Diarrhea, October 1923, gradually passing into constipation.

Roentgen-ray.—November 1923. Ulceration of proximal colon.

Two cases of diarrhea followed by diarrhea and constipation.

1. *Duration.*—Diarrhea two years, then gradually lengthening periods of constipation during the last two years.

Roentgen-ray.—Four series since May 1919, involving cecum, ceco-colon and first part of the transverse; March 1923, has almost entirely cleared up, only remaining evidence is complete emptying in twenty-four hours.

2. *Duration.*—Diarrhea for one month and then diarrhea and constipation for four months.

Roentgen-ray.—Involvement of cecum and ceco-colon.

One case of constipation followed by diarrhea and constipation.

Duration.—Constipation for a few weeks, diarrhea and constipation for six months.

Roentgen-ray.—Ulceration of the cecum.

Three cases of predominating constipation with occasional loose stools.

1. *Duration.*—Two years, pain severe lately.

Roentgen-ray.—Involvement of hepatic flexure region, constant in 4 series of plates (probably tuberculous).

2. *Duration.*—One year.

Roentgen-ray.—December 1923. Filling-defect of cecum and lack of haustration, small bowel probably involved.

3. *Duration.*—Six months, moderate constipation with loose stools at times.

Roentgen-ray.—November 1923. Cecum and first part of ascending colon involved.

One case of predominating diarrhea with occasional constipation.

Duration.—Six months, severe diarrhea preceded by a few days of constipation, recurring a few times.

Roentgen-ray.—Fairly definite but not wholly classical filling-defect of cecum.

Taking those cases in which considerable time has passed, we see that alternating diarrhea and constipation can continue for some time: in 3 cases for two years, in 2 cases for six months.

Taking the 4 cases in which there has been more than one roentgen-ray examination and in which it was shown in these roentgen-rays that there was definite improvement, we find that a real diarrhea developed with this improvement, as shown in the roentgen-ray in 2 cases. Thus, the development of diarrhea does not preclude an improvement of the lesion as shown on the films. In 1 case of these 4 the diarrhea and constipation continued. In 1 case gradually increasing constipation developed.

There seems to be nothing especially different in the roentgen-ray picture in these cases of diarrhea and constipation in the roentgen-ray picture, from those in the other cases of this series of 100, except in the 1 case with involvement of the hepatic flexure.

It is interesting to note that in only 3 of these 12 cases is an involvement of the small bowel revealed by the roentgen-ray. The alternation existed in 3 cases for two years, in 2 for six months. In 4 of these cases, a definite improvement was shown by the roentgen-rays and in 2 of these a real diarrhea developed. The alternation followed frank diarrhea in 3 cases, frank constipation in 3.

Nausea.—Nausea ranking next to pain and diarrhea in frequency, was present in 52 cases and was severe in 17, moderate or slight in 35. Thirty-five had vomiting at some time. Nausea never occurred alone as an initial symptom, but was accompanied with general digestive disturbances 18 times, vomiting 19 times, pain 14, general systemic disturbance 11, diarrhea 8 and constipation 4 times. It occurred after meals in 21, at any time in 12, in the morning in 8, at night in 5, with pain only in 3, and with menstruation in 1. It had persisted in 50 per cent of the cases for six months and in 5 for one year, lasting longer than pain but shorter than diarrhea. Over one-half the cases were accompanied by vomiting.

TABLE X.

	No. of cases.	Character	
		Severe.	Moderate or slight.
Primary period	31	12	21
Secondary period	14	4	8
Tertiary period	7	1	6
	52	17	35

Vomiting.—Vomiting was present in 37 cases, occurring almost always with nausea, which at times was slight. At times it was due no doubt to coughing.

TABLE XI.

	No. of cases.	Character	
		Severe.	Moderate or slight.
Primary period	20	9	11
Secondary period	14	6	8
Tertiary period	3	0	3
	37	15	22

General Digestive Disturbances.—General digestive disturbances, often referred to as indigestion, were present in 45 patients. They embraced the following:

Sour stomach	20
Distress after eating	28
Acid eructations, water brash, etc.	14
Heartburn	3
Fulness after eating a little	3
Bilious attacks with slight jaundice, headaches, etc.	1

In 5 cases, similar disturbances had existed from eight months to eight years or longer, and probably were only intensified by the intestinal tuberculosis.

They are more prominent as an initial symptom than diarrhea

or nausea, but in the long run are less important. They are nevertheless important as a suggestive symptom.

TABLE XII.

	No. of cases.	Character	
		Severe.	Moderate or slight.
Primary period	40	12	28
Secondary period	5	1	4
Tertiary period	2	1	1
	47	14	33

General Systemic Disturbances.—In 5 cases, elevation of temperature, out of proportion to the pulmonary sumptoms, was the outstanding symptom at the onset of the disease. In 17 other cases it was prominent and highly suggestive. In 11 cases, the presence of fever led to the roentgen-ray diagnosis of the intestinal tuberculosis.

In 15 cases, nervousness was prominent initially, but all had other more suggestive symptoms and 8 had previously had a similar nervous condition.

Table XIII contains the figures that are shown on the various charts of the duration of the individual symptoms. As in the charts the cases have been excluded in which the symptoms are still present, for in those cases it is, of course, impossible to tell how long these symptoms will go on. Some cases, however, in which the duration has been many months or years are included. These exceptions are indicated on each chart.

TABLE XIII.—DURATION OF MAIN SYMPTOMS.

	1 mo. or less.	2 mos.	3.	4.	5.	6.	7.	8.	9.	10.	11.	1 yr.	1 yr. to 2 yrs.	2 to 3 yrs.	3 to 4 yrs.	5 yrs. or more.
Pain	11	5	2	9	2	3	3	..	1	..	1	5	2	4	2	2
Diarrhea	6	4	3	9	2	4	3	2	2	1	1	3	11	4	2	2
Constipation	3	1	..	1	1	1	5	1	1	..	..	..	4	..	1	2
Nausea	5	3	3	9	4	3	5	1	2	1	1	6	1	2	1	1
General digestive disturbance	1	1	2	3	2	2	2	3	3	..	..	4	1	4	..	4

NEGATIVE CASES.

Erickson next proceeded to study 40 cases that had had symptoms so suggestive of intestinal tuberculosis that it had been deemed necessary to exclude it by roentgen-ray study. The figures given are percentages.

Onset.—The insidious or slight onset was present in 62.5 per cent and included slight diarrhea, 20 per cent; slight pain, 12.5 per cent; general digestive disturbances, 15 per cent; constipation, 7.5 per cent;

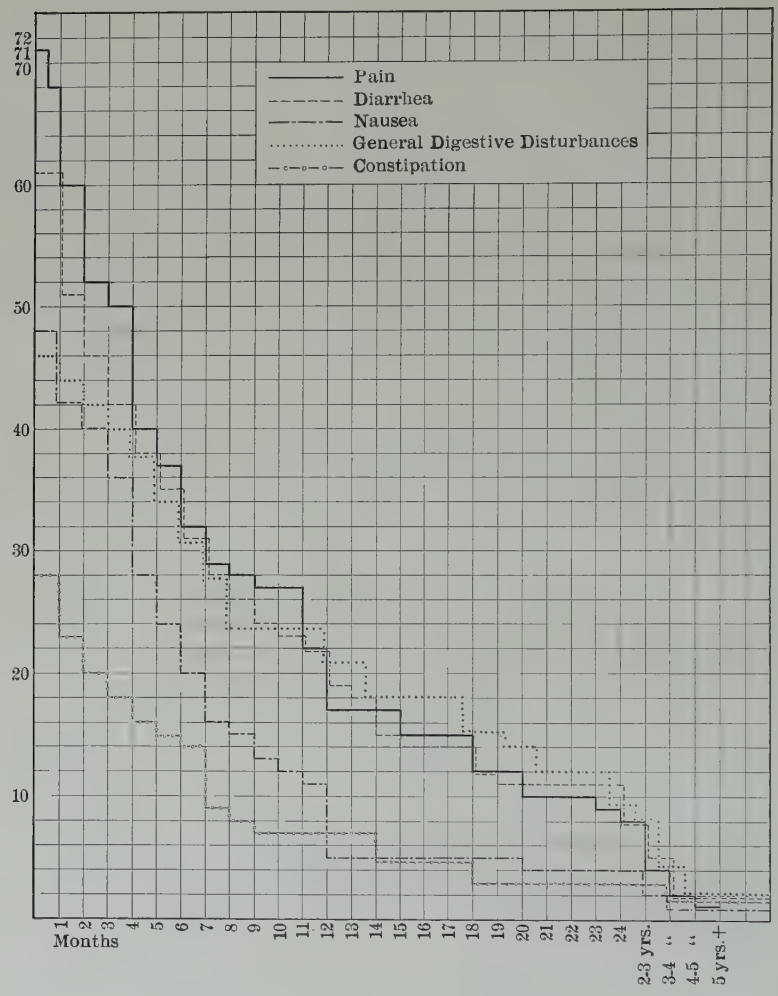

FIG. 17.—Duration of symptoms.

slight nausea and vomiting, 5 per cent; constitutional symptoms 2.5 per cent. The onset was predominantly with pain in 17.5 per cent, with diarrhea in 12.5 per cent and with nausea and vomiting in 7.5 per cent.

Symptoms.—Pain occurred in 52.5 per cent, constipation in 52.5 per cent, diarrhea in 45 per cent, nausea and vomiting in 35 per cent, general digestive disturbances in 37.5 per cent and predominating constitutional symptoms in 5 per cent.

It is of interest to compare the occurrence and character of the individual symptoms with those occurring in intestinal tuberculosis. Thus pain was present in the:

TABLE XIV.

	Negative cases, per cent.	Positive cases, per cent.
In	52.5	In 71.0
Severe	10.0	50.0
Mild	42.5	21.0

Pain was located in the:

	Negative cases, per cent.	Positive cases, per cent.
Right lower quadrant	12.5	31.0
Diffuse	10.0	21.0
Upper abdomen	15.0	10.0
Epigastrium	10.0	0.0
Lower abdomen	10.0	7.0

The character of the pain was in the:

	Negative cases, per cent.	Positive cases, per cent.
Cramping	20.0	46.0
Tenderness	5.0	6.0
Diffuse, vague	5.0	5.0
Aching	2.5	6.0
Burning	2.5	
Dragging	2.5	
Stabbing, sharp	5.0	5.0

TABLE XV.

A comparison of diarrhea and constipation follows:

	Diarrhea.		Constipation.	
	Negative cases, per cent.	Positive cases, per cent.	Negative cases, per cent.	Positive cases, per cent.
Present	45.0	67.0	10.0	28.0
Severe	15.0	....	2.5	9.0
Mild	30.0	....	7.5	19.0

Time of occurrence of diarrhea in:

	Negative cases, per cent.	Positive cases, per cent.
Morning	10.0	4.0
After meals	5.0	29.0
No special time	15.0	24.0
Night	2.5	10.0
Bleeding and diarrhea	5.0	13.0
Alternation of diarrhea and constipation	9.0	12.0

The occurrence of nausea and vomiting was as follows:

	Negative cases with vomiting, per cent.	Positive cases with vomiting, per cent.
Nausea present	35.0	52.0
Severe	5.0	17.0
Moderate or slight	30.0	35.0

General digestive disturbances were as follows:

	Negative cases, per cent.	Positive cases, per cent.
Present	37.5	48.0
Severe	0.0	14.0
Moderate or slight	37.5	31.0
The type:		
Distress or fulness after eating	25.0	28.0
Disturbance of appetite	12.5	26.0
Sour stomach	10.0	20.0
Food eructations	7.5	

General constitutional disturbances were as follows in:

	Negative cases, per cent.	Positive cases, per cent.
Present	7.5	11.0
Fever*	6.0	11.0
Nervousness*	6.0	15.0

* Apparently not explainable by pulmonary condition.

The following three tables, showing the percentages of the occurrence of the initial symptoms and the duration of the symptoms in the positive and negative cases, are of considerable interest:

TABLE XVI.—INCIDENCE OF SYMPTOMS IN POSITIVE AND NEGATIVE CASES.

	Total per cent occurring.		Per cent occurring alone initially.		Per cent occurring initially.		Per cent occurring secondarily.		Per cent occurring tertiarily.	
	Pos.	Neg.	Pos.	Neg.	Pos.	Neg.	Pos.	Neg.	Pos.	Neg.
Pain	71.0	52.5	0.0	5.0	39.0	37.5	25.0	15.0	7.0	0.0
Constipation	28.0	12.5	9.0	10.0	19.0	10.0	4.0	2.5	5.0	0.0
Diarrhea	66.0	45.0	6.0	5.0	36.0	32.5	19.0	12.5	11.0	0.0
Nausea and vomiting	52.0	35.0	3.0	5.0	31.0	30.0	14.0	5.0	7.0	0.0
General digestive disturbances	47.0	37.5	5.0	5.0	40.0	27.5	5.0	10.0	2.0	0.0
Constitutional symptoms	17.0	7.5	2.0	5.0	17.0	7.5				

TABLE XVII.—CONCURRENCE IN PERCENTAGES OF INITIAL SYMPTOMS IN POSITIVE AND NEGATIVE CASES.

	Pain.		Constipation.		Diarrhea.		Nausea and vomiting.		General digestive disturbance.		Constitutional symptoms.	
	Pos.	Neg.	Pos.	Neg.	Pos.	Neg.	Pos.	Neg.	Pos.	Neg.	Pos.	Neg.
Pain	..	..	4	0.0	17	17.5	14	15.0	13	17.5	3	25.0
Constipation	4	0.0	..	..	..	..	4	0.0	8	25.0	5	0.0
Diarrhea	17	17.5	0	0.0	..	..	8	7.5	13	7.5	8	0.0
Nausea and vomiting	14	15.0	4	0.0	8	7.5	..	..	18	7.5	11	2.5
General digestive disturbance	13	17.5	8	2.5	13	7.5	18	7.5	..	..	13	0.0
Constitutional symptoms	3	2.5	5	0.0	8	0.0	11	2.5	13	0.0		

TABLE XVIII.—DURATION OF DIGESTIVE SYMPTOMS IN POSITIVE AND NEGATIVE CASES.

	Pain.		Constipation.		Diarrhea.		Nausea and vomiting.		General digestive disturbance.	
	Pos.	Neg.	Pos.	Neg.	Pos.	Neg.	Pos.	Neg.	Pos.	Neg.
1 month	11	12.5	2	0.0	6	25.0	8	0.0	2	5.0
2 to 3 months	8	7.5	1	2.5	8	2.5	6	17.5	11	7.5
4 to 6 months	18	7.5	2	2.5	13	4.0	16	7.5	9	7.5
7 to 12 months	14	12.5	7	0.0	10	3.0	16	2.5	10	7.5
Over 1 year	13	0.0	7	7.5	23	1.0	10	2.5	21	10.0

In this table cases in which symptoms are still continuing, if of less than two months duration, are not included.

The following comparisons and conclusions were drawn by Erickson from his study:

"The symptomatology of intestinal tuberculosis is most varied. It is impossible to make out any one diagnostic syndrome or any one sequence of symptoms. The various symptoms occur and follow each other in manifold variety.

"Those cases, that were proved positive by the roentgen-ray, stood out from the negative cases, more by the multiplicity, and the severity and the duration of their symptoms than by any difference in kind or frequency of appearance.

"Positive cases are likely to have a definite onset twice as often as the negative cases. The insidious onset, though, is present in a third of the negative cases and, as such, cannot be differentiated from the same onset in the positive cases.

"Pain is a much more prominent symptom during the illness of the positive case than of the negative. When present at the onset, it is much more likely to be sharp and decisive in character and

definitely more likely to be localized in the right lower quadrant. Pain of less than one month's duration occurred twice as frequently among the negative as among the positive cases. Pain persisting over one year occurred only in the positive cases.

"Diarrhea is a more prominent symptom in the positive than in the negative case. At the onset, though, mild diarrhea is the most prominent symptom of the negative case. Thus, the diarrhea, that does not clear up soon or is of a severe character, is one of the most suggestive symptoms of a positive case. Diarrhea, of more than one month's duration, occurs six times as frequently in the positive as in the negative cases.

"Constipation at the onset is about as common in the negative as in the positive case, but in the positive case is likely to disappear, so that, as the disease goes on, it assumes a smaller and smaller part in the total picture. Thus, if it continues for a long time, it should suggest some other etiology. On the whole constipation shows no marked difference in the two groups.

"General digestive disturbances hold a somewhat smaller place in the onset of the positive than of the negative case. As the disease goes on, however, they are as frequent in the one as in the other, but, when they persist for a year they are twice as frequent in the positive as in the negative cases.

"Nausea at the onset occurs as often in the one type as in the other. As the disease goes on, however, it is more common in the positive than in the negative case. Vomiting with the nausea is decidedly much more frequent in the positive than in the negative case. Nausea, persisting for more than three months, occurs twice as frequently in the positive as in the negative cases, but, when it lasts for six months, it is four times as frequent in the positive as in the negative cases.

"Fever and general constitutional symptoms, that are not due presumably to the pulmonary disease, especially, if combined with suggestive though slight intestinal symptoms, are far and away more frequent in the positive case. In a limited number of cases it is thus of considerable value diagnostically.

"In conclusion, the positive case is characterized by the more severe type of onset, by the more acute symptoms and by the much greater variety, number and duration of these symptoms."

CHAPTER XVI.

CLINICAL EXAMINATIONS OF INTESTINAL TUBERCULOSIS.

FECES IN INTESTINAL TUBERCULOSIS.

The stools in intestinal tuberculosis vary. At times, especially in early cases, they are normal in appearance. As the disease progresses they become first mushy, then soft and little formed, and, finally, very watery, with a fetid, more or less characteristic, penetrating odor, due to the albumin, pus and peptone found in them. The liquid stool may be pale, due to larger fat and fatty soap content, or dark in color, at times frothy and may contain, besides particles of undigested and unabsorbed food, mucus, blood, pus, even shreds (sloughs) of the mucous membrane (rare), and much fat, due to the rapid peristalsis and extensive ulceration. Friedrich Mueller, in a child aged four years and in a woman aged thirty years with intestinal tuberculosis, found 40 per cent and 32.9 per cent, respectively, of the dried stool to be fat. Winternitz in an advanced case with amyloid disease found 32.7 per cent of fat in the stools, while in a normal person, under the same conditions, only 10 per cent was present. Macroscopic blood in the stools is seen more frequently when the rectum is affected but fatal cases of hemorrhage occur when the rectum is normal (see p. 219).

When a patient is on a mixed diet, bacteria compose 4 to 10 per cent, by weight, of the dried feces. The jejunum and upper part of the ileum are relatively bacteria-free. The more the motility of the intestine is disturbed, the greater amount of unchanged carbohydrate passes into the large intestine, and so more surely occurs stormy fermentation, producing a feeling of fullness and hindering the further emptying of the stomach, so that the fermentative processes are increased in this way, for the gas pushes up the diaphragm and the contents of the stomach become more acid (Durig). (See p. 136 for laboratory examination of stools.)

A careful chemical study of the stools of 17 patients with pulmonary and intestinal tuberculosis and of 13 with only pulmonary tuberculosis, made in the Trudeau laboratory by Mr. Frank Cooper, Chemist to the Trudeau Foundation, revealed no essential differences between the two groups (see Tables XIX and XX).

TABLE XIX.—CHEMICAL ANALYSIS OF FECES—NEGATIVE CASES.

Sanatorium No.	Color.	Odor.	Consistency.	Formed.	Reaction, pH.	Phenol.	Indol, Mg/gm.	Skatol, Mg/gm.	Fat, Mg/gm.	Stage.	Intestinal.
1	Olive-brown	Skatol	Putty	+	7.1	1.720	0.0148	0.0850	103.50	M. A. A.	Mineral oil.
4	Brown	Skatol	Mushy	−	7.4	1.504	0.0122	0.1475	79.40	M. A. B.	
7	Mahogany	Pungent	Pasty	−	6.9	3.170	0.0162	Trace	198.50	M. A. A.	Mineral oil daily.
9	Dark-brown	Pungent	Putty	+	7.0	1.560	0.0227	Trace	45.30	M. A. A.	Stools loose.
10	Golden-brown	Sour	Pasty	−	6.9	1.760	0.0058	None	155.80	M. A. A.	Mineral oil and cascara daily.
11	Olive-brown	Pungent	Pasty	−	7.4	2.570	0.0126	None	31.40	M. A. A.	
14	Dark-brown	Sour	Putty	+	7.1	1.605	0.0451	0.0089	57.10	T. B. ?	
15	Olive-brown	Pungent	Mushy	+	7.4	2.140	Trace	0.0099	46.60	M. A. A.	
17	Mahogany	Pungent	Doughy	+	7.0	2.970	0.0298	Trace	59.10	Min. A.	
19	Buff	Pungent	Stringy	+	7.2	2.099	0.0055	0.0180	149.36	F. A. A.	Mineral oil, 1 oz. daily.
20	Fawn	Sour	Doughy	+	7.3	0.894	0.0058	None	72.54	Min. A.	
21	Buff	Pungent	Pasty	−	7.6	1.872	0.0114	Trace	47.82	M. A. A.	

TABLE XX.—CHEMICAL ANALYSIS OF FECES—POSITIVE CASES.

Sanatorium No.	Color.	Odor.	Consistency.	Form.	Reaction, pH.	Phenol.	Indol, Mg/gm.	Skatol, Mg/gm.	Fat, Mg/gm.	Stage.	Intestinal.
2	Chocolate	Pungent	Mushy	−	6.8	0.900	0.0038	Trace	13.40	F. A. A.	Phenolphthalein taken.
3	Olive-brown	Pungent	Putty	+	7.0	2.010	0.0192	0.1778	69.00	M. A. A.	
5	Muddy-brown	Pungent	Putty	+	6.7	1.680	0.0657	0.0051	120.60	F. A. A.	Mineral oil, 1 oz. daily.
6	Olive-brown	Pungent	Mushy	−	6.7	1.850	0.0588	0.0079	Lost	F. A. B.	G.-I. upset present.
8	Buff	Pungent	Pasty	+−	7.2	2.750	0.0119	0.0223	82.60	F. A. A.	Had G.-I. upset.
12	Mahogany	Pungent	Putty	+	7.6	1.490	0.0314	Trace	87.00	M. A. A.	
13	Brown	Skatol	Pasty	−	7.6	2.500	0.0164	None	35.50	F. A. A.	Milk of magnesia daily.
16	Olive-brown	Pungent	Stringy	+−	6.8	2.940	0.0113	None	52.01	F. A. A.	
18	Olive	Sour	Stringy	+	7.4	1.600	0.0123	0.0207	58.90	F. A. A.	
22	Light-brown	Pungent	Pasty	−	7.0	1.730	0.0233	None	27.96	F. A. A.	
23	Fawn	Putrid	Stringy	+−	7.6	1.344	0.0136	Trace	138.86	F. A. A.	Mineral oil, calcium lactate and milk of magnesia.
24	Mahogany	Sour	Pasty	−	7.2	1.349	0.0139	None	48.20	F. A. A.	
25	Light-brown	Stale egg	Watery	−	6.8	0.581	0.0197	None	12.24	F. A.	Diarrhea.
26	Golden-brown	Pungent	Curdled Milk	−	7.1	0.791	Trace	None	29.90	F. A.	Diarrhea
27	Olive	Pungent	Mushy	+	7.6	1.456	0.0130	None	83.20	F. A.	Diarrhea.
28	Buff	Skatol	Creamy	−	7.8	0.507	0.0117	0.0050	17.77	F. A.	Diarrhea.
29	Dark-brown	Pungent	Pasty	−	7.1	1.495	0.0047	0.0035	133.40	F. A.	Mineral oil, 1 oz. daily.

TABLE XXI.—POSITIVE INTESTINAL ROENTGEN-RAY—NO PULMONARY CAVITY. (ROUTINE.)

Case No.	Date.	Hemoglobin.	Red blood. cells.	White blood cells.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Eosinophiles.	Basophiles.	Date of intestinal roentgen-ray.	
1	9–18–20	100.0	5,860,000	10,000	67.5	21.0	5.0	6.5	....	....	Nov., 1920	
	12–27–20	100.0	5,310,000	7,750	60.0	16.0	16.5	7.5				
2	1–12–22	120.0	.....	14,000	65.6	23.0	4.6	6.0	...	0.6	April, 1921	10 mos. after lamp.
3	10– 7–20	75.0	4,850,000	11,250	67.0	21.0	4.0	7.0	...	1.0	Oct., 1920	
	12–23–20	100.0	5,540,000	11,800	57.0	29.5	10.0	3.0	...	0.5		
4	12–29–20	95.0	6,560,000	18,400	62.0	21.3	10.3	5.0	...	1.3	June, 1920	
	12– 9–22	....	.....	23,800	55.0	8.0	23.5	23.5				
5	12–29–20	75.0	6,230,000	12,450	69.6	13.0	7.3	10.0	...	...	Aug., 1920	
	1–25–22	....	.....	8,200	71.0	28.5	5.5	3.5	1.0	0.5		
6	1– 4–21	100.0	6,050,000	8,200	57.6	22.6	13.0	3.3	3.0	0.3	.....	Diagnosis confirmed by operation, Nov., 1920.
7	1– 5–21	95.0	6,250,000	15,650	71.6	22.0	2.6	2.3	1.0	0.3	July, 1920	Artificial pneumothorax.
	1–11–22	....	.....	12,300	60.0	30.5	4.5	4.0	0.5	0.5		
8	11–12–21	95.0	5,700,000	20,200	75.3	9.0	5.6	9.3	0.6	...	Sept., 1920	
9	2–18–21	100.0	5,610,000	6,800	75.0	12.3	7.3	2.3	2.6	0.3	March,1920	
10	2–23–21	100. +	7,480,000	13,300	74.0	10.0	11.0	2.0	2.0	1.0	April, 1919	
11	2–25–21	100.0	6,880,000	9,400	55.0	20.0	20.6	3.3	1.0	...	Oct., 1920	
12	6–23–21	87.0	6,350,000	17,900	74.5	9.5	2.5	12.0	1.0	0.5	June, 1921	Poikilocytosis.
	4–13–22	....	.....	12,100	68.0	26.0	1.5	2.5	2.0			
13	7– 7–21	105.0	6,310,000	8,700	62.5	27.5	3.0	3.5	3.0	0.5	April, 9121	Roentgen-ray treatment, June, 1921: No lung roentgen-ray.
14	7– 9–21	91.0	5,330,000	10,300	72.3	17.3	6.0	2.3	1.3	0.3	Sept., 1921	
	1–12–22	....	.....	7,800	66.5	21.5	7.5	2.5	2.0			
15	8–27–21	....	.....	15,400	....	....	....	....	...	...	July, 1921	Tuberculous pneumonia. Valvular heart disease.
	9–24–22	....	.....	10,200	70.0	14.0	5.0	10.0	...	1.0		
16	10–28–21	....	.....	5,200	67.0	24.0	3.0	3.0	1.5	1.5	July, 1920	

17	12– 6–21	....	.....	18,200	75.0	12.0	3.0	8.5	1.5	...	Feb., 1922	Attack resembling appendicitis.
18	1–11–22	....	.....	17,300	61.5	25.5	5.0	6.0	2.0	...	Oct., 1920	
19	1–31–22	....	.....	10,100	60.5	34.0	2.5	2.5	0.5	...	Jan., 1922	
20	1–31–22	....	.....	21,350	82.3	9.0	3.0	5.0	0.6	...	Jan., 1922	
21	3–31–22	....	.....	15,200	74.0	21.0	2.5	2.5	0.5	...	March,1922	
22	4– 5–22	....	.....	23,500	71.0	19.0	3.6	5.0	1.3	...	March,1922	
23	5–20–22	....	.....	7,700	68.0	22.5	5.0	3.5	1.0	...	July, 1922	
24	5–20–22	....	.....	14,200	69.5	23.5	1.5	2.0	3.5	...	May, 1922	
	4–17–23	....	.....	12,700	60.0	11.5	24.0	2.0	2.0	0.5		
25	12 23–22	....	.....	18,200	75.5	11.0	9.0	4.5	...	...	Dec., 1922	
26	1–14–23	....	.....	13,000	71.0	11.0	13.5	2.5	2.0	...	Jan., 1923	Tuberculous cervical adenitis.
27	8–26–20	....	.....	4,200	80.0	5.0	13.0	2.0				Acute dysentery.
	9– 4–20	....	.....	13,800	62.0	14.0	17.0	7.0	...	...	Sept., 1920	
	9–19 20	....	.....	15,500	67.0	21.0	5.0	7.0				
28	2– 7–21	75.0	5,980,000	14,000							1–19–21	
29	2–21–21	....	.....	33,630	83.3	9.0	1.0	7.3	0.0	0.6	3–21–21	
30	5–15–24	10.5	4,940,000	12,850	70.0	24.5	2.5	1.5	1.5	1.5	4–24–24	
31	1–16 22	....	.....	9,300	63.5	25.0	7.0	2.0	1.5	1.0	6–22–22	Had dysentery for months. Sunlight and $CaCl_2$.
Average		95.0	5,954,707	12,995	67.9	18.6	7.4	5.1	1.0	0.3		

TABLE XXII.—POSITIVE INTESTINAL ROENTGEN-RAY—NO PULMONARY CAVITY (SARANAC LAKE LABORATORY.)

Case No.	Date.	White blood cells	Polymorphonuclears.	Lymphocytes	Large mononuclears.	Transitionals.	Eosinophiles.	Basophiles.	Date of intestinal roentgen-ray.	
1	4–11–21	11,450	73.5	5.0	12.0	18.5	4.0	1.0	3–23–22	Negative intestinal roentgen-ray. Tuberculous colitis at operation.
2	11–12–22	10,200	73.5	11.0	3.0	12.5	....	....	?	
3	3–20–23	14,050	75.5	8.5	10.5	5.0	0.5	....	4–12–23	
Average		11,900	74.1	8.2	8.5	12.0	1.2	0.3		

TABLE XXIII.—POSITIVE INTESTINAL ROENTGEN-RAY WITH PULMONARY CAVITY. (ROUTINE.)

Case No.	Date.	Hemoglobin.	Red blood cells.	White blood cells.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Eosinophiles.	Basophiles.	Date of intestinal roentgen-ray.	
1	2–21–20	90.0	5,380,000	11,650	58.0	27.0	8.0	6.0	1.0	...	Jan., 1920	
2	3– 1–21	90.0	5,650,000	18,200	74.3	10.6	7.6	5.3	1.6	0.3	Feb., 1921	
	1–21–22	....	.....	15,900	73.0	18.0	3.5	3.5	2.0			
3	4– 4–21	68.0	5,040,000	6,650	81.0	9.6	4.6	3.0	1.6	...	April, 1921	
	1–14–22	....	.....	8,900	77.5	12.5	2.5	6.0	1.0	1.5		
4	5–11–21	95.0	6,490,000	10,700	68.0	13.6	5.0	10.6	2.3	0.3	July, 1919	
5	6–25–21	90.0	5,280,000	17,400	84.6	9.6	2.6	2.6	0.3	...	June, 1921	Acute nephritis.
6	6–30–21	70.0	5,010,000	12,900	74.6	15.0	1.6	6.6	2.0	...	Aug., 1921	
	2–20–22	65.0	.....	14,900	81.0	11.0	2.0	5.0	0.5	0.5		
	4–17–23	....	.....	13,600	71.0	18.5	9.0	1.0	0.5			
7	8– 6–21	75.0	5,830,000	12,100	74.0	13.0	2.0	10.0	1.0		Aug., 1921	
	4–10–22	....	.....	16,800	70.3	15.0	1.6	5.3	1.0			
8	9–30–21	....	.....	21,000	86.0	....	10.3	2.1	0.6	0.3	Oct., 1921	Artificial pneumothorax
	1–15–22	....	.....	35,800	90.0	....	4.0	2.0	4.0	...	.....	Extension in lung.
9	10–12–21	....	.....	6,200	70.3	20.3	4.6	2.6	1.6	0.3	Oct., 1921	
	1–14–22	....	.....	7,100	63.5	23.5	6.0	4.5	2.5			
10	1– 7–22	....	.....	20,200	78.5	6.0	9.5	6.0	....	...	Feb., 1922	
11	12– 1–21	....	.....	10,200	70.0	26.0	3.0	....	1.0			
	1–27–22	....	.....	25,000	71.3	13.6	4.0	9.3	0.6	0.9	Jan., 1922	
	3–30–22	....	.....	29,500	80.0	6.5	3.5	8.0	....	0.5		
12	3–31–22	....	.....	18,300	80.5	10.0	3.5	6.0	....	...	March,1922	

13	4– 6–22	....	.....	14,300	79.0	7.3	2.6	5.6	4.6	...	March,1922
	4–17–22	....	.....	18,200	74.0	13.5	8.0	2.5	1.5	0.5	
14	5–27–22	....	.....	13,750	83.0	8.0	3.0	5.0	1.0		
	11–30–22	....	.....	17,100	85.0	5.6	9.0	2.6	0.3	...	Dec., 1922
15	2– 8–23	....	.....	20,400	65.0	26.5	6.0	1.5	1.0	...	Feb., 1923
	2– 9–23	....	.....	16,600	64.0	18.0	16.0	2.0			
	4–17–23	....	.....	11,800	52.0	31.5	14.0	1.0	1.5		
16	3– 6–23	....	.....	15,800	62.3	20.3	11.6	3.0	2.6		
	12–10–23	....	.....	41,800	88.6	5.0	1.3	5.0	...	...	Sept., 1922
17	10– 5 20	....	.....	15,300	80.0	9.0	4.0	7.0	....	...	3– 4–20
18	2–16–21	100.0	5,680,000	16,800	82.3	9.3	1.3	5.0	1.3	0.3	4–26–21
19	2–17–21	80.0	5,490,000	14,900	70.3	10.3	13.3	5.0	2.6	0.3	2– 8–21
20	7–22–22	....	.....	20,600	86.3	10.3	0.6	0.3	2.0	0.3	5–19–22
											2– 5–24
21	6–30–23	....	.....	23,600	88.5	8.0	2.5	1.0	....	...	9– 5–23
22	6–17–21	....	.....	7,500	81.0	10.5	3.0	4.5	1.0	...	Aug., 1922
23	6–28–21	85.0	5,250,000	6,000	57.0	36.5	4.0	2.5	1.5	...	June, 1921
24	10–12–21	....	.....	14,450	68.5	19.5	5.25	5.75	1.0	...	10–10–21
	1–20–22	....	.....	10,000	64.5	22.0	2.0	8.0	3.0	0.5	
25	1–12–22	....	.....	13,200	66.5	23.0	3.0	7.0	0.5	0.5	June, 1921
26	2– 4–22	....	.....	19,800	73.0	16.0	2.5	7.0	0.5	1.0	Feb., 1922
27	4–18–22	....	.....	9,350	58.0	26.5	6.0	9.0	0.5	...	April, 1922
Average		82.0	5,010,000	16,030	76.1	15.0	4.9	4.7	1.1	0.1	

	Acute gangrenous appendicitis.

TABLE XXIV.—POSITIVE INTESTINAL ROENTGEN-RAY WITH PULMONARY CAVITY. (SARANAC LAKE LABORATORY.)

Case No.	Date.	Hemoglobin.	Red blood cells.	White blood cells.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Eosinophiles.	Basophiles.	Date of intestinal roentgen-ray.	
1	9–14–21	....	.....	11,650	67.25	13.5	12.5	4.25	1.25	1.5	6–27–21	
2	1–29–22	....	.....	15,100	81.0	8.0	6.0	5.0	0.0	0.0	2–28–22	
3	2– 4–22	....	.....	22,500	79.5	8.0	1.0	11.5	0.0	0.0	3– 1–22	
4	3–21–22	....	.....	22,200	82.0	11.0	2.0	3.0	1.0	1.0	2–17–21 3– 3–22	
5	6–21–22	....	.....	12,300	69.5	5.5	15.5	9.0	0.0	0.5	4–28–22	
6	7–28–22	....	.....	8,000	61.2	10.1	21.5	3.0	3.0	0.0	5–11–21	
7	8– 2–22	....	4,640,000	17,500	81.0	4.0	9.0	6.0	0.0	0.0	4– 8–22	
8	8– 2–22	60.0	5,000,000	11,950	54.5	21.5	18.5	1.5	2.0	2.0	6–26–23	
9	10–13–22	....	.....	13,700	82.3	5.3	9.6	2.0	0.6	0.0	3– 9–21	Tuberculous meningitis.
10	11–23–22	....	.....	20,100	88.0	3.5	3.5	5.0	...	...	3–21–24	
11	11–31–22	....	.....	17,100	85.0	3.0	9.0	2.6	3.0	0.0	12–12–22	
12	2–26–23	....	.....	13,100	62.5	19.0	13.0	4.0	1.5	...	3– 5–23	
13	3–24–23	....	.....	10,400	64.5	14.5	13.5	7.0	0.5	...	11–28–21	
14	4–21–23	88.0	4,600,000	12,200	64.0	20.0	10.5	2.5	3.0	...	12–27–21	
15	1–14–23	50.0	3,540,000	6,300	63.0	18.0	15.5	3.0	0.5			
	4–21–23	65.0	4,350,000	7,320	72.0	21.0	4.5	1.0	1.0	0.5	4– 9–23	Blood transfusion.
16	6–30–23	....	.....	16,430	76.5	17.5	4.0	1.0	0.5	0.5	9– 7–23	
17	12–27–23	....	.....	7,950	72.0	14.5	10.5	3.0	...	...	2–15–23	
18	4– 2–23	....	.....	12,300	73.0	15.5	8.5	2.5	0.5	...	1–22–24	
19	6–13–23	60.0	3,200,000	15,150	59.8	13.5	22.5	3.0	0.5	1.0	4–10–23–	Acute nephritis.
Average		65.0	4,221,700	13,663	71.9	12.3	10.5	4.0	0.9	0.3		

TABLE XXV.—NEGATIVE INTESTINAL ROENTGEN-RAY.—NO PULMONARY CAVITY. (ROUTINE.)

Case No.	Date.	Hemoglobin.	Red blood cells.	White blood cells.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Eosinophiles.	Basophiles.	Date of intestinal roentgen-ray	
1	11– 8–20	100.0	4,430,000	12,100	62.0	18.0	14.0	6.0	...	...	10–29–20	
2	11–18–21	125.0	6,890,000	13,000	66.0	26.5	2.5	4.5	0.5	0.0	12– 5–21	Acute sinusitis.
3	2–21–22	....	.....	14,400	74.5	14.5	5.5	4.0	1.5	...	3–24–21	
4	5–12–22	80.0	5,960,000	15,500	59.5	34.5	2.0	3.5	0.0	0.5	8– 1–22	
5	1– 8–23	....	.....	11,950	81.0	11.0	7.0	1.0	...	...	6–26–22	Migraine attack.
6	3– 6–23	....	.....	12,000	57.0	27.0	11.5	3.5	1.0	...	7– 6–22	
7	4– 7–23	....	.....	12,450	62.0	25.5	4.0	6.5	1.5	0.5	2–15–23	Myocarditis; paroxymal tachycardia.
8	4–18–23	....	.....	6,700	47.0	32.0	16.0	4.0	...	1.0	2– 5–23	Tuberculous kidney.
9	7–19–23	....	.....	12,850	71.0	16.0	7.0	3.0	2.5	0.5	8–18–21	Chronic appendicitis, operation later.
											4–30–23	
10	10–30–23	....	.....	14,600	64.0	30.0	5.0	1.0	...	...	11–14–23	Appendicitis.
11	9–12 21	....	.....	11,650	67.5	24.5	3.5	3.5	1.0	...	.....	Chronic appendicitis.
	9–22–23	....	.....	10,000								
Average		102.0	5,760,000	12,266	64.7	23.6	7.1	3.7	0.7	0.2		

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TABLE XXVI.—NEGATIVE INTESTINAL ROENTGEN-RAY—NO PULMONARY CAVITY. (SARANAC LAKE LABORATORY.)

Case No.	Date.	Hemoglobin.	Red blood cells.	White blood cells.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Eosinophiles.	Basophiles.	Date of intestinal roentgen-ray.	
1	4– 6–21	....	.....	10,500	71.0	11.5	12.0	5.5	0.0	0.0	10– 6–20	
2	5–12–21	78.0	3,926,000	13,300	66.3	10.6	19.3	4.3	1.6	1.0	5–10–21	
3	9– 1–21	....	.....	10,000	63.0	18.0	16.5	4.0	3.0	0.5	4– 6–21	
4	4–18–22	....	5,920,000	8,010	66.8	8.9	16.9	4.4	1.7	0.8	7– 7–21	
5	6–14–22	....	.....	4,320	36.0	6.5	48.0	8.5	....	1.0	11– 1–22	
6	6– 9–22	87.0	5,400,000	4,640	48.5	33.5	7.0	5.5	4.0	1.5	4–19–22	
7	7– 5–22	....	.....	25,000	85.0	13.0	0.0	2.0	0.0	0.0	7– 1–21	
8	8–14–22	....	.....	9,900	64.0	16.5	8.5	11.0	....	....	1–31–23	
9	11– 8–22	72.0	4,925,000	9,500	58.0	22.5	9.5	3.5	5.5	1.0	10–19–22	
10	1–30–23	79.0	5,450,000	6,640	46.75	26.5	14.5	5.5	6.0	0.75	1–29–23	
11	5– 5–23	....	.....	13,700	76.75	19.0	2.0	1.5	0.75	0.0	2– 2–23	
12	6–29–23	73.0	4,437,000	6,400	66.5	20.5	3.0	6.5	3.0	0.5	7–25–22	Pneumothorax. Gastric ulcer?
13	10–27–23	66.0	4,536,000	10,100	78.0	16.5	3.0	1.5	1.0	0.0	11– 5–23	
14	10–30–23	76.0	4,410,000	9,800	68.0	29.0	1.0	1.5	0.0	0.5	10–22–23	
15	10–31–23	84.0	4,830,000	9,800	63.0	20.5	12.0	3.5	1.0	0.0	3–22–23	Tuberculous spine.
16	11– 8–23	....	.....	4,350	42.0	36.0	11.5	10.0	0.5	....	9–20–23	
17	11–16–23	80.0	4,570,000	8,600	70.0	11.5	14.5	3.5	0.0	0.5	4–15–24	
18	12–27–23	....	....	15,500	66.0	14.0	16.5	2.5	1.0	....	12– 4–23	Pleural effusion.
	12–31–23	....	.....	13,600	58.2	21.3	12.9	4.9	2.2	0.4	.....	Pleural effusion.
Average		77.0	4,850,400	10,192	62.8	18.7	11.9	4.7	1.6	0.5		

TABLE XXVII.—NEGATIVE INTESTINAL ROENTGEN-RAY WITH PULMONARY CAVITY. (ROUTINE.)

Case No.	Date.	Hemoglobin.	Red blood cells.	White blood cells.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Eosinophiles.	Basophiles.	Date of intestinal roentgen-ray	
1	10–27–20	66.0	4,536,000	10,100	78.0	16.5	3.0	1.5	1.0	0.0	12–30 22 9– 5–23	
2	5– 4–21	....	.....	13,500	75.6	13.3	7.0	3.3	0.3	0.3	11–20–21	Tuberculous purulent pleural effusion.
3	11– 3–21	....	.....	13,400	81.5	14.0	1.5	2.0	0.5	0.5	11–19–21	Tuberculous salpingitis.
4	1–17–22	....	.....	10,500	81.6	8.6	6.6	2.6	0.3	0.0	3–31–22	Artificial pneumothorax.
5	8–25–22	....	.....	9,300	79.0	12.5	0.5	6.5	1.0	0.5	3– 7–23	Grippe.
6	1– 8–23	....	.....	16,000	64.0	19.6	5.0	10.0	1.0	0.3	7– 1–23	Tuberculous purulent effusion. Artificial pneumothorax.
7	9–23–23	....	.....	10,700	85.0	12.0	1.0	0.0	1.0	1.0	10–21–23	
8	2–28–21	100.0	6,040,000	12,000	63.0	18.0	6.6	11.6	0.6	0.0		
	10–21–21	....	.....	12,800	....	....	...	....	...	...	.....	Diarrhea.
9	2–12–21	100.0	6,170,000	20,500	81.3	4.3	8.3	4.0	1.6	0.3	1920	Symptoms of enteritis.
10	9–15–20	95.0	4,730,000	12,100	77.0	22.0						
	2–22–22	....	.....	12,500	69.5	16.5	10.0	3.5	0.5	0.0	March,1922	Tuberculous spine.
Average		90.0	5,369,000	12,783	75.9	14.3	4.5	4.3	0.8	0.2		

TABLE XXVIII.—NEGATIVE INTESTINAL ROENTGEN-RAY WITH PULMONARY CAVITY. (SARANAC LAKE LABORATORY.)

Case No.	Date.	Hemoglobin.	Red blood cells.	White blood cells.	Polymorphonuclears.	Lymphocytes.	Large mononuclears.	Transitionals.	Eosinophiles.	Basophiles.	Date of intestinal roentgen-ray
1	10– 8–21	....	.....	28,250	77.7	2.0	11.5	9.2	0.0	0.0	11–25–21
2	11–14–21	....	.....	17,150	84.0	5.0	5.0	6.0	0.0	0.0	3–20–22
3	2–27–23	....	.....	6,880	68.5	19.0	6.0	2.0	4.0	0.5	10–13–22
4	2–28–23	....	.....	11,700	69.0	8.5	16.5	5.5	0.5	0.0	5– 7–23
5	4–20–23	....	.....	11,500	68.5	15.5	9.0	3.5	3.0	0.5	2–26–24
6	12–15–23	....	4,680,000	11,750	75.5	16.5	5.0	2.0	1.0	0.0	11– 9–23
Average				14,805	73.9	11.1	8.8	4.8	1.4	0.2	

TABLE XXIX.—RÉSUMÉ.

		Hemoglobin.	Red blood cells.	White blood cells.	Polymorphonuclears.	Lymphocytes.	Mononuclears.	Transitionals.	Eosinophiles.	Basophiles.	Cases.	Counts.
I. T. +, Cav. 0	Routine	95.0	5,954,707	12,995	67.9	18.6	7.4	5.1	1.0	0.3	31.0	42.0
Tables XXV, XIX	S. L. Lab.			11,900	74.1	8.2	8.5	12.0	1.2	0.3	3.0	3.0
I. T. +, Cav. +	Routine	82.0	5,010,000	16,030	76.1	15.0	4.9	4.7	1.1	0.1	27.0	42.0
Tables XXVI, XXII	S. L. Lab.	65.0	4,221,700	13,663	71.9	12.3	10.5	4.0	0.9	0.3	19.0	20.0
I. T. 0, Cav. 0	Routine	102.0	5,760,000	12,266	64.7	23.6	7.1	3.7	0.7	0.2	11.0	12.0
Tables XVIII, XX	S. L. Lab.	77.0	4,850,400	10,192	62.8	18.7	11.9	4.7	1.6	0.5	18.0	19.0
I. T. 0, Cav. +	Routine	90.0	5,369,000	12,783	75.9	14.3	4.5	4.3	0.8	0.2	10.0	12.0
Tables XXIV, XXI	S. L. Lab.			14,805	73.9	11.1	8.8	4.8	1.4	0.2	6.0	6.0

It is of interest to note that it was very difficult to find patients, whether in the Trudeau Sanatorium or in the village of Saranac Lake, suffering from chronic diarrhea due to intestinal tuberculosis. An examination of smears from these specimens, made by Dr. S. A. Petroff, showed the presence of acid-fast organisms in but one specimen, from a patient with both pulmonary and intestinal tuberculosis. Little difference in the bacterial flora between the two groups was noted.

THE BLOOD IN INTESTINAL TUBERCULOSIS.

The blood, in rare instances, may give a picture suggesting pernicious anemia, due possibly to strictures in the small bowel and absorption, but usually the most characteristic finding is a leukocytosis, which is unusual in uncomplicated pulmonary tuberculosis without cavity formation.

In a study of the blood, Lemon found the average hemoglobin percentage was 66, the lowest reading 30, the highest 85, using Dare's hemoglobinometer. The average erythrocytes were 4,150,000, the lowest count 2,700,000, the highest 5,400,000. The average leukocytes were 8975, the highest 19,400 and the lowest 3600. There were 12 patients with counts of 11,000 or more among the 48 cases in which counts were made.

J. N. Hayes, in his study of 80 cases of secondary ulcerative intestinal tuberculosis diagnosed in our laboratory, found 46 had and 34 had no pulmonary cavity. He further counted the blood cells of 45 patients without intestinal tuberculosis, 16 of whom had pulmonary cavities. The figures are given in tables XXI to XXIX, together with their averages, pages 138 to 146. While the number of counts is small, a study of the mean and the mode both point to an essential increase in the leukocytes, due to intestinal tuberculosis. When a pulmonary cavity is present, intestinal tuberculosis does not seem to influence the differential count, which is little affected, whether or not intestinal tuberculosis coexists. When intestinal tuberculosis is present, the coexistence of a pulmonary cavity may slightly increase the number of polymorphonuclear and consequently decrease the number of mononuclear cells present. He deemed it wise to separate the cases in which he made routine counts, from those made in the Saranac Lake Laboratory, because in the latter some complication was suspected.

THE BLOOD-PRESSURE.

In his study of the blood-pressure, Lemon noted a low pressure in nearly all cases, which averaged 109/72, the lowest being 92/52 and the highest 125/90.

A study of the blood-pressure of 52 men and 45 women among our cases with pulmonary and intestinal tuberculosis showed the following averages:

TABLE XXX.

		No. of cases.	Systolic pressure.	Diastolic pressure.	Pulse-pressure.
Moderately advanced	Males	25	115	78	41
	Females	33	110	73	35
Far advanced	Males	27	112	71	40
	Females	12	108	71	38

Among these 97 cases there were:

TABLE XXXI.

	Systolic pressure.			Diastolic pressure.		Pulse-pressure.
	above 120.	above 130.	below 100.	above 80.	below 70.	above 50.
52 males:						
Moderately advanced 25	..	2	2	4	6	2
Far advanced 27	..	4	1	3	9	4
45 females:						
Moderately advanced 33	4	1	6	5	10	2
Far advanced 12	1	1	4	1	4	1
97 Total	5	8	13	13	29	9

The lowest was 88/46 in a male in a moderately advanced condition, and the highest was 144/90, also in a male but in a far advanced stage.

THE URINE.

Routine examination of the urine, for albumin, casts, sugar and cellular elements, showed that in 217 cases the urine was normal in 100 and that in the majority of the remainder only slight or temporary changes were present.

One patient developed an acute nephritis from which she made a good recovery. The table on p. 149 shows at a glance the findings. The cases in the village of Saranac Lake are characterized by a greater severity, and it is of interest to note the more frequent occurrence of albumin among them. The occurrence of sugar in 5 of the Trudeau cases was noted but on a single examination in each case and was very slight in amount. It is of interest to note that a waxy cast was found but once. The urochromogen test, a routine procedure at Trudeau, was positive only once and then negative on six subsequent examinations.

TABLE XXXII.—URINARY FINDINGS.

A. In 107 Trudeau cases:

	Male.	Female.
Normal	38	19
Abnormal	18	32
Pus abnormal quantity		
On one examination only	5	
Only one examination	..	4
On many examinations	..	5
On 2 or more examinations and then normal	..	11
R.B.C. on one examination only	1	2 (1 with acid-fasts; guinea-pig negative).
Casts:		
On one examination only:		
Hyaline	4	1 (with pus).
Hyaline and granular	3 (1 with acid-fasts; 1 with pus and R.B.C).	1
Granular	1	3 (1 with pus; 1 with sugar).
Blood	..	1
On many examinations:		
Hyaline and granular	2 (1 with trace of albumin).	
Hyaline	2 (1 with pus; 1 with trace of sugar, subsequently normal).	
Sugar	..	5

(One with trace of albumin and few granular casts, 2 with pus.) One with hyaline casts.

B. In 110 cases in Saranac Lake Village:

	Male.	Female.
Normal	22	21
Abnormal	38	29
Abnormal quantity of pus	7	2
Pus and albumin	2 (1 with R. B. C.)	7 (T. B. +, guinea-pig in 1).
Albumin	15 (1 with R. B. C.)	12 (T. B. +, guinea-pig in 1; 1 with R. B. C).
Albumin and casts:		
Hyaline	1 (with pus and R. B. C).	3 (1 with few pus cells).
Hyaline and granular	3 (1 with R. B. C.)	
Granular	4	4
Casts	1 (with few R. B. C. and few pus cells).	
Casts alone:		
Hyaline	1	
Granular	2 (1 with pus).	
Waxy	1	
Acute nephritis	..	1 (good recovery).

A study of the urine by Walsh showed 65 per cent of his cases with ulceration had casts or albumin (or both) and indican, and 27 per cent a diazo-reaction, while 80 per cent of those without ulceration had casts or albumin or both; 61 per cent had indican and 20 per cent a diazo-reaction.

CHAPTER XVII.

THE RELATION OF SYMPTOMS OF INTESTINAL TUBERCULOSIS TO THE SITE OF THE LESION.

This subject has been studied by many observers, who have attempted to associate certain symptoms with the various pathological changes found at autopsy. It must constantly be borne in mind that it has not always been easy to say, when the ulceration first occurred in certain segments of the intestine.

It is of interest to recall that Louis in his careful work noted that the diarrhea was less copious in patients in whom only ulceration and no softening of the mucous membrane had occurred. In 15 patients with remittent diarrhea, ulceration was present in the small bowel in 10, in the large in 6; chronic and continued diarrhea was present in 41 patients, 35 with ulceration in the small, and 31 in the large bowels. In these cases the ulcerations were more extensive, intensive, numerous, and apparently of longer standing, than in the group with remittent diarrhea. If the large ulcerations were confined to the small bowel, the diarrhea was no less chronic and continuous, than if confined to the large intestine, where such changes were considered the chief cause of such diarrhea.

This subject has been very carefully studied by Archibald in his excellent paper. When ulcers occur in the small bowel, constipation is often marked but in a few cases diarrhea has been noted. The pains, due to gas, are transient, crampy, not severe, generalized and felt irregularly here and there. Anorexia may be troublesome, while slight distention, flatulence and occasional nausea may occur. When such symptoms persist, when they are relieved by fasting and no thickening of the cecum can be made out, tuberculous enteritis (*i. e.*, tuberculous ulceration of small bowel) should be thought of. When such a pain, as has just been described, occurring at intervals, localizing itself over the cecum, coming on only or chiefly in the afternoon, two or three hours after meals but disappearing at night, and followed by soreness, is associated with a palpable cecum, ulceration, at least of the cecum and possibly involvement of the appendix and colon must be considered. In such cases constipation occurs early, then alternates with diarrhea, and finally

two or three loose stools a day occur. Anorexia may be early and persistent. The transverse and descending colon are silent areas. When, as so often occurs, both large and small bowels are involved, Archibald believes, any or all of the above conditions may be present.

Kieffer holds that in his cases extensive ulceration in the small bowel is usually accompanied by diarrhea, not constipation. Engelsmann, in his 100 autopsied cases noted stubborn diarrhea in 44. He thinks when diarrhea occurs with isolated disease in the small intestine that some inflammatory condition is also present in the large bowel. He found that diarrhea always accompanied ulceration in the large bowel.

Schwatt, in an interesting study of 127 ulcerated cases, found only 49 presented symptoms, referable to the intestinal tract. He has classified these cases according to the pathological lesions found at autopsy in relation to the various symptoms. In 30 cases, where the ulceration was limited to the small intestine, 22 presented no symptoms either before admission or during the period of observation. In 20, the bowels were regular or there was slight constipation, and in 2, constipation was severe. Diarrhea was a prominent symptom in 8. In 6 of these, the ileum alone was diseased, in 4 of which, diarrhea alternated with constipation.

He thinks that the character of the lesions and the extent of the disease in the intestine, or the degree of involvement of the peritoneum has little to do with the symptomatology.

In 18 cases with the disease limited to the large intestine, those with the disease limited to the cecum appear to run a latent course. In 1 case, with diarrhea one month before exitus, there were a few recent small ulcers in the cecum and a chronic passive congestion of the jejunum and ileum.

In 7 cases of ulceration of the cecum and colon, with or without disease of the appendix and rectum, diarrhea was present in 6, colicky pain in 5, tenderness to palpation in 2 and rigidity in 2.

In 20 cases with involvement of the small intestine and cecum, with and without involvement of the appendix, 12 ran a latent course, with at times slight constipation. Marked constipation existed for several months before death in 1 case with numerous advanced ulcers of the ileum and cecum.

Diarrhea was present in 6, colicky pain in 7, tenderness to palpation and rigidity in 4, cecal tumor in 2, blood in the stools in 1. In 1 case with advanced ulceration of the lower ileum, cecum and appendix, the bowels were always regular, and a diagnosis was made

on severe abdominal pain, tenderness to palpation and rigidity. In 1 case only with a severely ulcerated appendix, the pain was always localized at McBurney's point.

The involvement of the appendix seems to increase the number of symptoms. The degree and extent of the disease of the ileum and jejunum had apparently no bearing on the symptomatology. The same is true in regard to the cecum.

In 56 cases of ulceration of the small and the large intestine, only 24 presented symptoms referable to the intestinal tract, that is, diarrhea in 19, colicky pain in 20, tenderness in 13, rigidity in 10, palpable tumor in 1 and blood in the stools in 1. In this group, Schwatt found, diarrhea occurred in a considerably greater number of cases of ulceration involving both the jejunum and ileum, 14 out of 30, than when the ileum alone was involved, 6 out of 25. The severity of the disease of the cecum and ascending colon apparently had no relation to the occurrence of this symptom, but diarrhea was more likely to occur, when a large number of segments of the colon were involved. Ulceration of the rectum apparently has the same bearing on the presence of diarrhea.

Lemon, in his excellent study of intestinal tuberculosis, publishes an interesting table of his 50 cases, which follows:

TABLE XXXIII.

	Alternating constipation and diarrhea	Diarrhea.	Constipation.	Normal or not stated.	Percentage.
Pain in the abdomen	4	13	14	14	90
Described as cramping or as colic	1				
Cramping	..	4	5	2	
Burning	..	1	1		
Epigastric	..	4	1	5	
Food distress	..	..	2		
Located in epigastrium	..	..	1		
Mid-abdomen	..	..	1		
Typical appendicitis	..	..	..	1	
Right lower quadrant	..	..	..	5	
Tender abdomen, soreness	3	5	5	1	28
Upper	..	..	2		
Lower	..	..	3		
Tumor palpable	1	3	5	5	28
Right lower quadrant	..	..	4		
Palpable stricture	..	..	1		
Nausea	2	..	3	3	16
Vomiting	3	2	7	4	32
With relief	..	..	1		
Digestive disturbances	4	10	14	3	62
Flatulence	..	10			
Gas	4	10	7	1	
Distention	4	10	2		
Belching	4	10	1		

TABLE XXXIII—(*Continued.*)

	Alternating constipation and diarrhea	Diarrhea.	Constipation.	Normal or not stated.	Percentage
Digestive Water-brash	4	10	..	3	
Food distress	4	10			
Loss of appetite	..	10	1		
Sour stomach	..	10	3		
Ascites	..	1	..	..	2
Night-sweats	..	1	..	..	2
Weakness	..	2	4	1	14
Hemorrhage	..	2	..	..	4
Nervousness	..	..	2	..	4
Stools, mucus	..	..	5	2	14
Blood	..	..	..	1	
Melena	..	..	..	1	
Lesion:					
Jejunum	..	..	1	1	4
Ileum	3	6	7	11	54
Ileo-cecal valve	..	..	3	..	6
Appendix	2	4	2	2	20
Cecum	3	8	12	9	64
Colon	1	5	3	5	28
Transverse colon	..	2	..	4	12
Ascending colon	..	6	..	..	12
Descending colon	..	..	..	1	2
Rectum	..	..	1	1	4
Glands	1	4	5	3	26
Fistula	..	1	1	2	8
Peritoneum	2	7	1	2	24
Miliary	2	1	1	1	10
Mesentery	..	..	..	1	2
Fissure-in-ano	..	1	..	..	2
Tubes	..	1	..	..	2
Omentum	1	..	..	..	2

The view, held by some writers, that the location and extent of the ulcerative lesions do not agree with the symptoms, is true in many cases, but unlike the work of Archibald, is based only on autopsy findings, where the disease is often widespread. It is often impossible to define accurately the extent of the intestinal lesions by roentgen-ray examination. Tenesmus occurs usually only when the rectum is involved.

As a matter of fact, the whole subject is in a most unsatisfactory state. Conclusions, drawn from conditions existing at autopsy, are not altogether satisfactory but, on the other hand, at operation certain lesions may be overlooked. Unfortunately, this condition holds in regard to many other conclusions about intestinal tuberculosis and has been unhappily, but unavoidably, the cause of retarding progress.

CHAPTER XVIII.

THE ABDOMINAL EXAMINATION IN INTESTINAL TUBERCULOSIS.

The abdomen, early in the course of the disease, may be normal in every particular of outline and on palpation. Archibald, however, thinks that in cecal involvement careful palpation usually reveals some thickening. Later, of course, distention or, at times, retraction of the abdomen is present. In the later stages, definite thickening and, at times, a definite mass can be palpated, most often in the right lower quadrant. Tenderness may, of course be present over this or other areas without intestinal tuberculosis, but is more frequent and often more pronounced when it is present.

In his 45 cases, Pritchard found slight rigidity in 1, and a palpable mass in 2, one of which was a hypertrophic type of intestinal tuberculosis. Tenderness was noted in 22, most frequently in the right lower quadrant. A palpable mass and tenderness were both present in 14 of Lemon's 50 cases.

A sharp definite pain on pressure, which may radiate to the epigastrium or to the left, is due, some think, to definite involvement of the peritoneum over the base of the ulcer. Hyperesthesia of the skin may occur. The referred pain may be found in the skin, muscles or connective tissue, which receive their innervation from the same segments of the spinal cord (sixth to twelfth), which in turn, receive the afferent stimuli, passing from the intestinal tract through the splanchnics (Pottenger).

Increased muscle tone, which has proved so valuable in the diagnosis of simple appendicitis, should be valuable also in tuberculosis of the intestines, Pottenger thinks, especially in those patients, who are in good physical condition with normal abdominal muscles. If, however, this infection should be established later, after degeneration of the muscles has occurred as a result of the disease, then increased muscle tonus would be extremely difficult to determine with any degree of certainty.

Pigmented areas may appear on the hands and face, and some have described pigmentation of the ulcers and of the overlying peritoneum. Fistula occurs more often in connection with intestinal tuberculosis, but Walsh found it only 8 times in 76 cases and once in 24 cases of pulmonary tuberculosis without intestinal tuberculosis.

CHAPTER XIX.

THE PULMONARY CONDITION IN SECONDARY INTESTINAL TUBERCULOSIS.

The pulmonary condition, in the majority of cases of secondary intestinal tuberculosis, is advanced, which, of course, is to be expected, for usually not until a great part of the patient's resistance is lost, do symptoms of intestinal tuberculosis become apparent. In fact so common is intestinal tuberculosis in far advanced stages that, from the time of Hippocrates until today, the incontestable symptoms of intestinal tuberculosis have been held to point in the majority of cases to a speedy demise. Schwatt says death occurs usually within three to six months. But a different state of affairs comes to pass in those patients with only moderately advanced pulmonary disease. Here are to be found many of the remittent type, many whose intestinal tuberculosis is discovered only because some keen observer is on the lookout for it, and suspects it from symptoms, passed over by many good clinicians, who expect intestinal tuberculosis to be associated only with the terminal stages of pulmonary tuberculosis. Or again, it may be discovered only on routine examination and the patient be practically free from symptoms.

Intestinal tuberculosis is very rare in our experience among the patients in the minimal stage, but that some do suffer from it is confirmed by our studies. The view, advanced by Lebert and later by Stewart and Pritchard, that the intestinal tuberculosis may be the primary disease, is not borne out by our figures, for among 65 patients in the minimal stage examined at Trudeau, none had intestinal tuberculosis. In 316 cases of secondary ulcerative intestinal tuberculosis, only 5 occurred in patients in the minimal stage, and it is questionable if some of these should have been placed here. Certainly the majority of all cases arise only in the more advanced stages of pulmonary tuberculosis, for in the group studied, 171 occurred among patients in the moderately advanced stage and 138 among those in a far advanced stage. It is highly probable that secondary intestinal tuberculosis arises as frequently as, or indeed more frequently, in patients with moderately advanced pulmonary

tuberculosis, than it does in those with far advanced disease. The pulmonary disease after the onset of intestinal tuberculosis, may, however, rapidly advance. This emphasizes again the great importance of diagnosing the complication at a time, when the patient has not lost all his resistance and can still respond to suitable treatment.

In Table XXXIV are found the pulmonary condition of 107 patients in the Trudeau Sanatorium with both pulmonary and intestinal tuberculosis and 209 similar cases studied in the village of Saranac Lake. It seems advisable to separate the figures into these two groups, for the types of cases are very different. Only the more favorable cases are admitted to the Trudeau Sanatorium, while in Saranac Lake severe cases are frequent. This is borne out in part by the figures, but they fail to tell the whole story, for many of the far advanced cases in Trudeau demand a far better prognosis than a large number of the moderately advanced cases in the village.

TABLE XXXIV.—CONDITION OF LUNGS IN PATIENTS WITH SECONDARY INTESTINAL TUBERCULOSIS.

No. of cases.	Stage.	Male.			Female.		
		No. of cases.	Pulmonary cavity.		No. of cases.	Pulmonary cavity.	
			Present.	Absent.		Present.	Absent.
	A.	*At the Trudeau Sanatorium*					
	Minimal	0	0	0	0	0	0
79	Moderate advanced	42	18	24	37	15	22
28	Far advanced	15	8	7	13	7	6
107		57	26	31	50	22	28
	B.	*In Saranac Lake Village*					
5	Minimal	2	0	2	3	0	3
93	Moderate advanced	58	10	48	35	6	29
111	Far advanced	52	43	9	59	47	12
209		112	53	59	97	53	44
316		169	79	90	147	75	72

The more frequent occurrence of pulmonary cavities in the village cases would suggest also that their disease was more severe It should be borne in mind that the occurrence of a cavity, larger than one interspace, automatically places the case in the far advanced group. It is then easy to see that no minimal case exists

with a pulmonary cavity and that, in the moderately advanced stage, only cases with beginning cavity formation occur. Among the moderately advanced cases only 49 (28 per cent) out of 173 cases had pulmonary cavities, while they occurred in 105 (76 per cent) of 138 far advanced cases. On the whole, cavity formation occurs in about 48 per cent of all the cases, but it must not be forgotten that many of these cavities can be diagnosed only by means of the roentgen-rays. The cases have been classified solely on their pulmonary condition and the occurrence of laryngeal, intestinal and other complications has been disregarded.

Cavity formation seems a trifle less frequent among the female patients but the figures are small and the difference, neither marked nor greatly significant.

THE OCCURRENCE OF TUBERCLE BACILLI IN THE SPUTUM IN PATIENTS WITH SECONDARY INTESTINAL TUBERCULOSIS.

Of the 277 cases studied, 151 were males and 126 females. Among them, 137 cases (49 per cent) were without and 140 (51 per cent) with a pulmonary cavity. Of the 152 moderately advanced cases, 106 (69 per cent) had none and 44 (31 per cent) a cavity, while of the 125 far advanced cases, 31 (25 per cent) had none and 94 (75 per cent) a cavity. Of all the cases, 25 (9 per cent) had no tubercle bacilli, and, among the cases with pulmonary cavity, only 2 were bacilli-free (1.4 per cent). A study of the number of bacilli is of some interest. Among 68 moderately advanced cases without pulmonary cavity, 63 per cent had Gaffky VI (7 to 12 tubercle bacilli to the field) or less, and 37 per cent over Gaffky VI; while for the far advanced the figures were 72 per cent and 28 per cent respectively. After the development of a cavity, for the moderately advanced the figures were 56 per cent and 44 per cent, and for the far advanced 47 per cent and 53 per cent. Of the 86 cases without pulmonary cavity, 65 per cent had Gaffky VI or under and 35 per cent over Gaffky VI, while for those with cavity (107) the figures were the same for each group (50 per cent). It can be readily seen that the cases with pulmonary cavity have, as a rule, a larger number of tubercle bacilli in the sputum, which, of course, was to be expected. The most striking point of the whole study is that these cases do not show more tubercle bacilli in their sputum and that there is so little difference between the cases with and without pulmonary cavity. These figures are based upon a study of Table XXXV.

TABLE XXXV.—TUBERCLE BACILLI IN SPUTUM OF CASES OF SECONDARY INTESTINAL TUBERCULOSIS.

			Cases without pulmonary cavity.					Cases with pulmonary cavity.				
			No. of cases.	T. B. ○	T. B. +	Gaff. VI & under	Over Gaff. VI	No. of cases.	T. B. ○	T. B. +	Gaff. VI & under	Over Gaff. VI
Trudeau cases.	Moderate advanced	Male	24	7	—	11	6	18	1	—	10	7
		Female	22	5	..	11	6	15	0	—	5	10
	Far advanced	Male	7	0	..	2	5	8	0	—	1	7
		Female	6	2	..	4	0	7	1	..	3	3
Village cases.	Moderate advanced	Male	40	6	14	10	10	7	0	2	4	1
		Female	30	2	14	11	3	6	0	2	4	0
	Far advanced	Male	8	0	7	1	0	39	0	8	17	14
		Female	10	1	3	6	0	40	0	19	10	11
			147	23(16)	38	*56(65)	*30(35)	140	2(1)	31	*54(50)	*53(50)
		Moderate advanced	116	20(17)	28	43(63)	25(37)	46	1(2)	4	23(56)	18(44)
		Far advanced	31	3(10)	10	13(72)	5(28)	94	1(1)	27	31(47)	35(53)
		Male	79	13(16)	21	24(53)	21(47)	72	1(1)	10	32(52)	29(48)
		Female	68	10(15)	17	32(78)	9(22)	68	1(1)	21	22(48)	24(52)

*The percentages in parenthesis in these columns refer only to cases in which the number of bacilli are noted. "Gaffky VI" indicates seven to twelve bacilli to the field.

CHAPTER XX.

DIAGNOSIS OF INTESTINAL TUBERCULOSIS. CLINICAL METHODS.

The diagnosis of intestinal tuberculosis has undergone a revolutionary change within the last decade. The newer methods are apparently so little understood, so feebly grasped, by many excellent clinicians, that, without question, many patients now succumb to tuberculosis who otherwise might be saved. It has appeared wisest to discuss first the older clinical methods of diagnosis and then to deal fully with the newer roentgenological diagnosis, pointing out, that the latter method alone can give an accurate idea of the presence or absence of secondary ulcerative intestinal tuberculosis.

The diagnosis of secondary intestinal tuberculosis from symptoms and physical signs is notoriously uncertain and many gastroenterologists have until recently been very reluctant to make a positive diagnosis of intestinal tuberculosis, even in the presence of more or less active pulmonary tuberculosis.

The position of intestinal tuberculosis in the minds of many specialists, even today, is similar to that occupied by pulmonary tuberculosis fifteen or twenty years ago. At that time, many refused to make a diagnosis of pulmonary tuberculosis without finding tubercle bacilli in the sputum, regardless of physical signs and symptoms, and the struggle to overcome this reluctance was long and hard. At the beginning of this century, the presence, in a patient with advanced pulmonary tuberculosis, of chronic diarrhea, of more or less abdominal pain, of tender points in the abdomen, with or without rigidity, and of nothing suggestive of an acute abdominal condition, led to the diagnosis of intestinal tuberculosis and a uniformly fatal prognosis. In a few instances, patients placed on treatment, for example, with small doses of creosote and iodoform, lost their abdominal symptoms, which did not recur up to the time of death. Such cases were regarded by conservative thinkers as probably mistakes in diagnosis.

A careful study of 100 cases autopsied by Engelsmann, showed that a diagnosis had been made in only 33; of the 67 cases without a diagnosis, 35 presented no clinical symptoms and yet 23 had

severe intestinal tuberculosis, 1 a caseous and 1 a purulent peritonitis. One of the 35 had lost nearly all the mucous membrane of the large bowel. In the other 32 cases symptoms were found on going over their histories after the autopsies, but they were considered during life too slight for diagnosis. Diarrhea was present in 19, constipation in 11, pain in 7, tenderness on pressure in 6. Diarrhea was the only symptom in 11, in 9 of which the disease was extensive and in 2 confined to the ileo-cecal region. Constipation was present in 10, 2 with general disease and 8 with local (2 in the small bowel and 6 in the ileo-cecal region). Constipation occurred early or late. In 33 cases with a positive diagnosis, diarrhea occurred in 25, constipation in 5, pain in 15, tenderness on pressure in 7, distention in 4, gas in 6. As the only symptom, diarrhea occurred in 7 and gas in 1.

As a rule, the more severe the case proved to be, the greater were the number of symptoms. Over two-thirds of the whole series were not diagnosed.

In a study of 452 cases with intestinal tuberculosis at autopsy, Kieffer had diagnosed 250 clinically. The remaining 42 per cent showed no characteristic symptoms. In about 150 of the cases the intestinal tuberculosis was slight.

Walsh, in a careful study of 100, autopsied cases at the Phipps Institute (Philadelphia), aged from nine to fifty-seven years at death, found tuberculous ulceration in the large or small intestine 76 times. On comparing the positive and negative cases he found the following:

TABLE XXXVI.

Intestinal ulceration.	Diarrhea.	Pain.	Abd. tend.	Abd. rig.	All 4 present.	All 4 absent.
Present 76 .	31 (40.8%)	21 (28.0%)	23 (30.3%)	19 (25.0%)	5 (6.58%)	26 (34.2%)
Absent 24 .	7 (29.2%)	7 (29.2%)	7 (29.2%)	6 (25.0%)	1 (4.2%)	9 (37.5%)

From these observations, he concluded that it is impossible to diagnose accurately the presence or absence of intestinal tuberculosis and, furthermore, that the fear of the effect of a positive diagnosis upon patient, nurse and physician, which he has so well described, and the fact that so bad a prognosis has always accompanied such a diagnosis, should deter us from making it in the interest of the patient.

"A clinical diagnosis of tuberculous ulceration of the intestine cannot" says Schwatt in 1922, "as has been shown above, be made

in the majority of cases . . . In our 127 cases the intestinal complication was recognized in 49 or 36.8 per cent.

"Although the presence of tuberculous ulcers in the intestine," he continues, "cannot be ruled out by the absence of symptoms, it may be said, with a negligible degree of error, that diarrhea in far-advanced phthisis, especially in cases with laryngeal ulceration, becoming more and more persistent and uncontrollable, practically always justifies a diagnosis of this complication. If, in addition, there are colicky pain and meteorism, the diagnosis is certain" (see p. 24). In the great majority of his cases he noted the onset of symptoms only three to six months before death. There can be no doubt that Schwatt has repeatedly overlooked many early symptoms of intestinal tuberculosis.

Diarrhea may, therefore, occur without intestinal ulcers and tuberculous ulcers without diarrhea, which, as has already been stated, depends largely upon the condition of the intact mucous membrane and to a less degree upon the location of the ulceration. An ischio-rectal abscess should always direct attention to the possibility of the presence of intestinal tuberculosis. Persistent localized pain is always very suggestive. The occurrence of unchanged bilirubin and of unabsorbed remnants of food would indicate hypermotility of the small bowel.

Intestinal tuberculosis remains, therefore, today as difficult to diagnose by clinical methods as it has always been, and we must pursue other lines, if we wish to escape such pitfalls and to prescribe for our patients appropriate and early treatment. (See p. 229).

ABDOMINAL EXAMINATION.

A careful abdominal examination may reveal some thickening of the cecum or in a few instances in later stages, a mass in the right lower quadrant. In passing it should be noted that not only carcinoma, hypertrophic cecal tuberculosis, but amœbiasis and actinomycosis can also produce masses in this region. In this last condition the sulphur-yellow granules have been found in the stools (Ransom). On the other hand, ulceration of the ileum, cecum and colon may and frequently does occur without thickening of the intestine. Tenderness is more often present when the ileo-cecal region is involved but tender points may occur elsewhere. Some believe that marked abdominal tenderness or rigidity is a definite sign that the peritoneal surface over the base of the ulcer is involved.

CHAPTER XXI.

DIAGNOSIS OF INTESTINAL TUBERCULOSIS (*Continued.*) LABORATORY METHODS.

Failing by clinical means to make an accurate diagnosis, many have studied various laboratory procedures, leading to the discovery in the stools of the presence of tubercle bacilli, of blood and of pus.

TUBERCLE BACILLI IN THE FECES.

The source of the tubercle bacilli, that are found in the feces of many patients with pulmonary tuberculosis, has been long and widely discussed. At first, and in fact even today, some hold that they come from the tuberculous intestinal ulcers and always indicate intestinal tuberculosis. Many studies have shown that such a position cannot be maintained, and most tubercle bacilli come undoubtedly from the swallowed sputum. More recently, attention has been called to the fact that tubercle bacilli may be excreted in the bile.

1. **The Sputum as the Source of Origin.**—Lichtheim, in 1883, was the first, it may be recalled, to demonstrate tubercle bacilli in the stools, where he found them only a little less numerous than in the sputum. He held, however, that tubercle bacilli in the stools occurred only in patients with intestinal tuberculosis. Craemer a little later found what he considered tubercle bacilli in the stools, not only of patients suffering from pulmonary tuberculosis and diarrhea, but also in the stools of well and sound persons. But Marchand and others who examined Craemer's slides found them improperly decolorized. De Giacomi thereupon studied the stools of 50 sound persons and found no acid-fast bacilli, which, however, were always present in the feces of patients, who at death showed intestinal ulceration. Demme and Kredel also upheld this view, *i. e.*, that tubercle bacilli in the feces were a sign of intestinal tuberculosis. Gaffky, at Koch's suggestion, studied the feces of sound persons and found no tubercle bacilli.

Bodo, in 1891, examined at autopsy the stools and intestinal contents of 9 patients, dead of pulmonary tuberculosis. Three

had advanced intestinal tuberculosis but he found no tubercle bacilli in their intestinal contents or feces. In 3 others without intestinal tuberculosis, he found tubercle bacilli (in 1 in large numbers) in both feces and intestinal contents. In 3 other cases with intestinal tuberculosis, he found tubercle bacilli in 1 only in the intestinal contents, in the other 2 in both the intestinal contents and feces.

Biermann, in 1896, also advanced the view that tubercle bacilli in feces were of no great value in the diagnosis of intestinal tuberculosis.

It has long been questioned whether or not acid-fast organisms found in the feces were really tubercle bacilli. Mironescu in 1901 isolated from the stools an acid-fast organism, possibly the butter bacillus, with slight animal pathogenicity. Jousset, in 1903, obtained an acid-fast organism from the stool of 2 patients with typhoid fever, which was not a tubercle bacillus. Ferrán isolated acid-fast rods from the feces of man and cattle and thought they were a form of colon bacillus, related to the tubercle bacillus.

Strasburger found an acid-fast organism, morphologically not a tubercle bacillus, in the feces of 2 non-tuberculous persons. Schroeder and Cotton were never able to find non-tuberculous, acid-fast organisms in the rectal scrapings of non-tuberculous cattle nor could McFarland and Beardsley discover them in the feces of rabbits and guinea-pigs. Briscoe and MacNeal frequently found acid-fast bacteria which were not tubercle bacilli in the feces of cattle that failed to react to tuberculin.

Bodo's and Biermann's work, however, was overlooked and various methods were devised to facilitate the search for tubercle bacilli in the stools as a means of diagnosis of intestinal tuberculosis.

Rosenblatt used opium to condense the stools for examination, and Strasburger developed a technic that enabled even solid stools to be studied successfully for tubercle bacilli. He mixed a bit of the stool, the size of a pea, with 1 cc. of water, centrifuged gently, poured off the supernatant fluid, added to it twice its volume of alcohol, 96 per cent, and centrifuged vigorously.

Anglade and Chocreaux, in 1902, found tubercle bacilli almost always in the feces of patients with intestinal tuberculosis and thought them of diagnostic significance.

Landis, in 1907, found tubercle bacilli in the stools of 13 patients, but, at autopsy, only 9 had tuberculous intestinal ulcers.

Klose, in 1910, examined 60 patients with tubercle bacilli in the sputum and found them 55 times in the feces. In only 6 of these

was there any suggestion of intestinal tuberculosis. In 12 controls, without tubercle bacilli in the sputum, he found none. He added 25 cc. of 50 per cent antiformin to a pea-sized mass of feces, and shook often for twenty-four hours. He then added 60 cc. of water and 2 to 3 cc. ligroin, shook vigorously and then stained the intermediate layer. Englesmann found no tubercle bacilli in the stools of 7 patients, 4 of whom had intestinal tuberculosis. In 33 cases with tuberculous ulcers at autopsies, the stools had been negative in 5. Bergstrand, in 60 non-tuberculous persons, found no acid-fast organisms in the feces.

In 1910, Philip and Porter attempted to combat the view that tubercle bacilli in the feces meant intestinal tuberculosis. They studied the feces of 99 patients with pulmonary tuberculosis and 1 with a non-pulmonary form. Of the 100 cases, 75 had acid-fast organisms in the stools including 33 of the 34 which had them in their sputum. Of 42 patients with pulmonary tuberculosis and tubercle bacilli in the sputum, 29 had acid-fast bacilli in the stools, while of 24 patients with pulmonary tuberculosis and no tubercle bacilli in the sputum, 17 had acid-fast bacilli in the stools. In 9 presumably healthy persons, no acid-fasts were found. They were unable to identify the acid-fast organisms, either by culture or by animal inoculation.

During the same year, Alexander found acid-fast organisms in the stools of 52 of 81 patients with tuberculosis (in 38 of the 45 with pulmonary tuberculosis). He inoculated guinea-pigs with the feces of 24 advanced cases and 23 developed tuberculosis. He found no acid-fast organisms in the stools of 129 non-tuberculous individuals.

Rittel-Willenko, in 1911, examined 54 patients and found tuberculous ulcers in nearly all who had tubercle bacilli in their feces. In 22 patients with acid-fasts in the sputum, she found them only 3 times in the stools and concluded that tubercle bacilli in the feces were an indication of intestinal tuberculosis, but few now agree with her.

In 1913, Laird, Kite and Stewart studied at the Trudeau Sanatorium 153 patients with pulmonary tuberculosis. They found by staining methods, in a study of 54 patients without tubercle bacilli in the sputum that the feces were positive for acid-fast organisms in 2 and negative in 52. Of the patients with positive sputum, in 60 the feces were positive and in 40 negative.

After inoculation into animals of 87 specimens of feces containing acid-fast organisms, 48 guinea-pigs developed tuberculosis, 14

remained well and 25 died of other disease. Of 34 guinea-pigs inoculated with feces, containing on smear apparently no acid-fast organisms, 1 developed tuberculosis, 22 remained well and 11 died of other disease.

Schöne and Weissenfels, from their work in 1914, concluded that Reh's method was the best but suggested that instead of water 5 to 20 drops of a 50 per cent solution of antiformin be added to a piece of feces the size of a pea.

Engleson, in 1915, compared the various methods of searching for tubercle bacilli in the stools with his method of staining the rectal mucus removed by a blunt curette. He studied 60 cases, 7 in the first, 2 in the second and 51 in the third stage (Turban). His results are as follows:

TABLE XXXVII.

Tubercle bacilli in sputum.		Reh's method.		Rectal mucus.	
Positive.	Negative.	Positive.	Negative.	Positive.	Negative.
41 (68%)	19 (32%)	44 (73%)	16 (27%)	57 (95%)	3 (5%)

Engleson found Reh's method better than any other for direct examination of the feces. (A piece of feces the size of a bean, is mixed with enough water to make it half fluid. Ether is added and the whole shaken. The supernatant ether is poured into a tube and centrifuged. The sediment is then fixed on a slide and stained.)

From this, it would appear that a study of the rectal mucus should be made in all patients suspected of having pulmonary tuberculosis in whose sputum no tubercle bacilli can be found, which Sawyer advised in 1906 and Schroeder and Cotton used in animal work.

More recently (1923), B. M. Fried examined the feces for tubercle bacilli in 3 series of patients at Mattapan, Massachusetts.

TABLE XXXVIII.

	Tubercle bacilli in sputum.		Tubercle bacilli in feces.	
	Positive.	Negative.	Positive.	Negative.
1st series	103	0	98	3
2d series	0	11	3	8 (All clinically tuberculous.)
3d series	0	12	0	12 (All clinically non-tuberculous.)

In 20 of his 45 positive cases of pulmonary and intestinal tuberculosis, Pritchard (1923) found tubercle bacilli in the stools, 2 of which had none in the sputum.

Nüssel in 1923, examined the stools of 120 patients with pulmonary tuberculosis, 6 of whom had intestinal tuberculosis, and found acid-fasts, which he determined by staining methods to be tubercle bacilli in 110. He says all who have tubercle bacilli in the sputum have them in the stools and, if they occur in the stools and isolated intestinal tuberculosis be excluded, they will be found in the sputum. If intestinal tuberculosis be excluded, the number in the stools vary directly with that in the sputum. Ordinarily the usual direct smear will reveal the presence of tubercle bacilli in the feces, if they are present, but in a few antiformin may have to be used. His figures follow:

TABLE XXXIX.

Tubercle bacilli sputum.	No. cases.	Tubercle bacilli in stools.		Benzidin test after meat-free diet.		I. T. clinically present, tubercle bacilli in stools.		Stage Turban.			Pulmonary condition questionable.
		+	○	+	○	+	○	I	II	III	
+	120	110	10	..	120	6	0	1	28	91	0
○	120	0	120	..	..	..	..	32	13	3	72

Gant stated as late as 1923 that the presence of tubercle bacilli in the feces is the only proof of intestinal tuberculosis.

Loll performed an interesting experiment upon 2 robust patients with pulmonary cavities and tubercle bacilli in the sputum. He ordered them to cough up as little sputum as possible for one or two days. They of course swallowed most of it. The feces at the end of that time were loaded with tubercle bacilli, whereas previously, there were none or few found. He thinks the presence of tubercle bacilli in the feces of no value in the diagnosis of intestinal tuberculosis.

Sergent and Durand (1923) always found tubercle bacilli in the stools of 8 patients who had them in the sputum, but failed to find them in 13 non-tuberculous individuals and in 16 suspected cases, none of whose sputum, of course, contained tubercle bacilli. Venot and E. Moreau (1923) found tubercle bacilli in the stools of 41 of 42 patients with tubercle bacilli in the sputum, but failed to discover them in the stools of 35 patients with various affections, including some cases of tuberculous pneumonia, where the sputum was always

free from tubercle bacilli. These authors think patients are reinfected through the swallowed sputum. The technic used in these studies was as follows:

About 50 gm. of feces are placed in a vessel if fluid, or in a mortar and mixed with 25 per cent NaCl to make a thin emulsion, if solid. The mass is then filtered through gauze, and put in centrifugal tubes to which 2 cc. of a mixture of equal parts of ether and ligroin are added. The tubes are centrifuged at 4000 to 5000 revolutions per minute for ten minutes. The tubercle bacilli if present are found usually just under the ether-ligroin layers in a thin layer which can be used for staining and for injection of guinea-pigs.

Falci in 1923 studied the feces of 44 patients, all of whom had advanced pulmonary tuberculosis and 4 had symptoms of intestinal tuberculosis as well. Tubercle bacilli were found in 3 of these 4, but he says there was no definite relation between the number of tubercle bacilli in the sputum and the number in the stools. He modified the technic of Venot and Moreau, using ether-petroleum instead of ligroin, as it was cheaper.

2. **The Bile as the Source of Origin.**—The observations of L. Rabinowitsch, of Fraenkel and Krause, of Calmette and of Corper on the secretion of tubercle bacilli through the bile into the intestine may throw doubt also upon the importance in the diagnosis of intestinal tuberculosis of any tubercle bacilli found in the stools, but it must not be forgotten that tubercle bacilli coming from this source are found usually only by animal inoculation and not by direct smears.

Fraenkel and Krause, in 1899, in 36 cases of intestinal tuberculosis found the bile sterile by smear in 34, but, when the bile of 11 of these was injected into guinea-pigs, five became tuberculous. Rabinowitsch, in 1913, in a study of the bile of 11 subjects with almost generalized tuberculosis and intestinal tuberculosis, found tubercle bacilli in 3, after using antiformin. In these 11 and 5 other cases with intestinal tuberculosis, guinea-pig inoculation showed tubercle bacilli present in 12.

Corper found tubercle bacilli in the bile of 6 of 20 tuberculous cases at postmortem, of which 18 were far advanced.

Koizumi found tubercle bacilli in the bile in 50 per cent of the cases he studied in man, and believes the real figures are much higher.

Calmette and Guérin recovered tubercle bacilli in the bile of rabbits, which they had inoculated intravenously with tubercle bacilli two to four days previously. Calmette infected 15 of 109 guinea-pigs with the bile from a fistula in a tuberculous heifer, and 3 of 66 guinea-pigs with the bile from a second. Titze and Jahn

found tubercle bacilli by inoculation in the bile of about 25 per cent of hogs and cattle with generalized tuberculosis.

E. Libert inoculated subcutaneously a dog with a complete biliary fistula with a large dose of tubercle bacilli. No tubercle bacilli were found in the bile and attempts at growth on Petroff's media and inoculation of guinea-pigs were both negative, although the dog died on the thirty-eighth day from generalized tuberculosis. He thinks the bile in man, like the urine from a normal kidney, may contain occasionally a few tubercle bacilli.

It is clearly evident that the few tubercle bacilli in the bile have little chance of being found in the feces, as has been pointed out by Schöne and Weissenfels, who conclude that tubercle bacilli in feces practically always come from swallowed sputum. They mention a case with infiltration of the upper lobe, where, after the use of antiformin, only 2 or 3 bacilli were found in a field in the sputum and only 1 in 50 fields in the feces. Six weeks later masses of tubercle bacilli were found in the sputum and at the same time in the feces.

The frequency of tubercle bacilli in the stools of patients with pulmonary tuberculosis is suggested by the study of Brown, Petroff and Heise, who found viable tubercle bacilli in the water of a river as far as 3 miles below the mouth of a sewer from a health resort. Near the opening of the sewer the bacilli were very numerous. Tubercle bacilli were found as long as feces were evident macroscopically.

Petroff (1912) was the first to devise a method by which tubercle bacilli could be grown directly from the stools.

Sasano working in our laboratory was able to find tubercle bacilli in the bile of guinea-pigs and rabbits only when the liver was affected.

It must not be overlooked, however, that in patients without sputum or with bacilli-free sputum, the presence of tubercle bacilli in the feces would suggest intestinal tuberculosis. On the other hand, absence of tubercle bacilli does not exclude tuberculous ulceration, for it is doubtful if many tubercle bacilli are ever thrown off from the ulcers into the intestinal contents.

3. **Tubercle Bacilli in Feces of Cattle.**—The feces of tuberculous cattle have also long been known to contain tubercle bacilli. Schroeder and Cotton, in 1907, maintained that the chief source of tubercle bacilli in milk was its contamination with feces containing tubercle bacilli. By feeding 3 cows tubercle bacilli, they proved that passage through the alimentary tract did not destroy their virulence for guinea-pigs or hogs. In a study of 7 tuberculous cows, they found acid-fast organisms in 5, 3 of which were proved

by animal inoculation to be tubercle bacilli. Schroeder believes that 40 per cent of all tuberculous cows have tubercle bacilli in the feces, which usually come from the lungs and not from the intestinal mucosa, but tubercle bacilli are found only when the tuberculosis is open, *i. e.*, in cases of bronchial ulceration. Probably the 45 cattle examined by Reynolds and Beebe were almost all closed cases, for only 1 contained tubercle bacilli in the feces, though all reacted to tuberculin. Peters and Emerson had the same experience for they found tubercle bacilli in only 3 of 41 reacting cattle.

Six cows, which had contracted tuberculosis naturally, were found by the Royal Commission on Tuberculosis of Great Britain to be passing virulent tubercle bacilli in the feces.

Briscoe and MacNeal, in 1911, after a number of experiments found that a pure culture of tubercle bacilli in quantities as small as 100 billionths of a gram, suspended in a 1 per cent feces-emulsion, produced tuberculosis in guinea-pigs. They then examined 119 specimens of feces from 53 cows, reacting to tuberculin and found acid-fast bacilli 53 times in 34 cows. Twenty-eight per cent of the 53 reacting cows gave negative results. About 17 per cent of the feces of 18 non-reacting cows contained acid-fast bacilli. So acid-fast organisms that are not tubercle bacilli, may occur in the feces of cows, and many have concluded that the occurrence of acid-fast organisms in the feces is of little or no aid in the diagnosis of tuberculosis in cattle. These authors further examined 97 samples from 62 reacting cattle and found tubercle bacilli in the feces in only 3 (5 per cent).

Schroeder, in 1908, had pointed out, however, that while the tubercle bacilli were fairly evenly distributed throughout the feces, it may be very difficult to find them, and, in most instances, it was necessary to examine the feces daily for a week or more and in 1 instance for several weeks to discover tubercle bacilli, for in the earlier stages tubercle bacilli are excreted only intermittently. The acid-fast organisms were proved to be tubercle bacilli by inoculation of guinea-pigs and cattle. In a number of cattle examined, only 1 was found with tuberculous intestinal lesions, which might doubtfully be the source of the tubercle bacilli. On the other hand Pearson and Ravenal (1901) in a pathological study of 1200 tuberculous cattle found the small gut involved in 388 (32.5 per cent) and the large in 189 (15.8 per cent).

From this somewhat full statement of the relation of tubercle bacilli in the feces to the presence of secondary intestinal tuberculosis, it can easily be seen that, if looked for carefully enough, tubercle bacilli

can be found in 95 per cent or more of all cases with tubercle bacilli in the sputum and, furthermore, that the presence of tubercle bacilli in the feces is no indication whatsoever of the presence of intestinal tuberculosis, nor does their absence exclude it under such conditions. It is striking how few acid-fast organisms that are not tubercle bacilli are found in the feces of man. They have been described by Moeller, Ferrán, Mironescu, Severin, Olt, Capaldi and others.

4. **The Occurrence of Blood in the Stools.**—There is, of course, no more reason to expect to find blood in the stools of patients with intestinal tuberculosis than in the sputum of patients with pulmonary tuberculosis, and, as is well known, some cases of pulmonary tuberculosis even go on to extensive cavity formation and never show any macroscopic blood. Macroscopic blood is rather rarely observed in the feces (see p. 218) but opinions differ about the presence of minute traces.

Landis, in 1907, after controlling the diet, studied the stools of 32 patients, who later came to autopsy, for occult blood by the benzidin test. Of these, 6 had no tuberculous ulcers and in 5 to 7 tests each, never showed the presence of blood. In the other 26 cases, all with tuberculous ulceration, 12 were negative in 1 to 3 tests, and 1, indeed, in 6 tests. The other 14 were positive. The bleeding, of course, may be intermittent and he thinks if the test were repeated frequently enough more than 50 per cent would become positive.

Joachim, in studying many patients, has never obtained a positive test in any instance, where there were no intestinal ulcers.

Sahlgren, after carefully excluding from the feces all other sources of blood than the intestinal wall and after controlling all his reagents, studied the stools of 120 patients with minimal as well as advanced pulmonary tuberculosis, for occult blood by the benzidin, phenolphthalein and guaiac tests. The last he considers of little value. In 98 patients, the benzidin test was positive but only 52 had any intestinal symptoms and in only 17 were they pronounced. Forty-one of them came to autopsy, and 1, in whom the benzidin test was always negative, had no ulceration. Of these cases, only 24 had intestinal symptoms, which were pronounced in only 17. In 32 of the remaining 40 cases with tuberculous ulceration, the benzidin test was positive. All of the 8 negative cases had pronounced intestinal ulceration, which in 6 was extensive. The intensity of the benzidin test bears no relation to the extent of the ulceration, but, together with the occurrence of hemoptysis, seems

more closely connected with either an acute or chronic course (tendency to healing?). The occurrence of the occult blood apparently bears no relation to the presence of hard feces (scybala).

He found no cases of stenosis. In 7 of the 40 autopsies, the larynx was tuberculous. In only 5 was there a pronounced amyloidosis.

In concluding, Sahlgren states that the presence of occult blood is good evidence of the presence of intestinal tuberculosis, but that its absence is of no value in excluding such a condition. If the benzidin test becomes positive after the 4 to 8 days of strict diet, it is of less value in diagnosis than if, always positive, it increases in intensity.

Loll is a great believer in the benzidin test as a method of revealing intestinal tuberculosis. He has never found ulceration at autopsy, when this test was negative just before death. He prescribes a most careful diet and gives no food containing hemoglobin for six days and tests no stool until the fifth day. If it is negative three times, he says it will always be negative. In a study of 150 cases, he says the test was positive when ulceration existed. Kuttner, on the other hand, found that the test was often negative in far-advanced cases. Kirch, in a study of 27 cases, found 5 gave a negative benzidin test when tuberculous ulceration was present.

In 30 out of 42 specimens of feces examined from patients with intestinal tuberculosis, Pritchard found occult blood after a meat-free diet for three days.

There is apparently no definite relation between the extent of the disease and a positive reaction for blood, for extensive ulceration can exist without reaction. On the whole, a positive reaction occurs in intestinal tuberculosis more frequently than symptoms. In carefully controlled cases, a positive benzidin test is suggestive of tuberculous ulceration but a negative reaction is of no value in excluding this condition.

PUS IN DIAGNOSIS OF INTESTINAL TUBERCULOSIS.

Few routine examinations have been made for pus in the stools of patients with intestinal tuberculosis. Pritchard found it in 32 of his 45 patients but never in large amounts.

Soluble albumin, which, according to Coope, indicated ulceration, was found in 32 of his cases by Pritchard but was absent in some of the severer cases and was never marked.

Fiorito has obtained good results in the diagnosis of intestinal tuberculosis with Roncal's alizarin reaction with the intestinal mucus.

THE BLOOD IN THE DIAGNOSIS OF INTESTINAL TUBERCULOSIS.

The blood picture affords little aid in diagnosis, as the leukocytosis, which is often present, may be fairly attributed, as Hayes' work shows, in part to the presence of cavity formation in the lung, rather than wholly to the intestinal tuberculosis. But a high leukocytosis does suggest the possibility of intestinal tuberculosis, especially in the absence of pulmonary cavity and of other complications.

TUBERCULIN IN DIAGNOSIS OF INTESTINAL TUBERCULOSIS.

Tuberculin may cause some increase of abdominal pain, but aids little in diagnosis.

RECAPITULATION.

To recapitulate briefly, it seems likely that either slight rise in temperature without increase of pulmonary symptoms, anorexia prolonged and marked, slight digestive disturbances, regurgitation or vomiting of food, nausea, slight abdominal discomfort or actual pain, constipation or slight diarrhea, constipation alternating with diarrhea, or an unexplained leukocytosis should attract attention to the intestinal tract and suggest possible tuberculous ulceration. It must always be borne in mind that the presence of several of these symptoms usually occurs only when the process is well marked. In fact, any patient with chronic pulmonary tuberculosis, who fails to gain weight or to make satisfactory progress, no matter how slight the abdominal symptoms are, should be studied for latent tuberculous colitis. The frequency of such symptoms in the course of pulmonary tuberculosis is well recognized, but we hold that in the presence of such symptoms a diagnosis of intestinal tuberculosis cannot be made positively, except by roentgen-ray study.

CHAPTER XXII.

THE DIAGNOSIS OF INTESTINAL TUBERCULOSIS (*Continued*). ROENTGEN-RAY METHODS.

It has been shown by what has already been presented upon the subject of the diagnosis of intestinal tuberculosis that, before the roentgen-ray examination of the opaque meal was satisfactorily worked out, the diagnosis of this condition was often suspected when not present, and frequently overlooked even though extensive ulceration of the bowel existed. It is hoped that the method, described in the following pages, will enable others, as it has the authors, to diagnose intestinal tuberculosis in its earliest stages, or, indeed, as soon as ulceration has occurred. It is equally useful for advanced cases. In fact when pseudo-filling-defects are excluded, it is incontrovertible evidence of an ulcerative bowel condition. It seems hardly necessary to note that it diagnoses only ulceration and affords no information about the cause of the ulceration. In other words the differential diagnosis of the various ulcerative conditions of the small and large bowel must hinge upon additional data. In patients with pulmonary tuberculosis, the presumptive evidence is so strongly in favor of intestinal tuberculosis that other conditions require usually but little thought.

We have deemed it wise to describe in considerable detail the normal outline of the bowel as revealed by a study of an opaque meal and enema, believing, that some, who we hope will use the method we are now to describe, are not familiar with the normal. We have attempted to go into many details, apparently trivial to roentgen-ray experts, but, at times, possibly unfamiliar to students of pulmonary tuberculosis, upon whom in the end the roentgen-ray examination of the bowels will largely fall. We would like to emphasize again the importance of the section on pseudo-filling-defects, for only by excluding these can frequent mistakes be avoided.

NORMAL OUTLINE OF THE GASTRO-INTESTINAL TRACT.[1]

The study of the outline of the gastro-intestinal tract can be made possible in two ways: (1) By filling it with some radiopaque sub-

[1] It has been deemed wise to repeat here in part what has already been stated in previous chapters.

stance such as barium or bismuth; or (2) by the introduction of gas. The former, being more practical and in many ways superior, is the method more commonly used. The administration of the radiopaque material in suspension is either by meal (barium or bismuth meal) or by enema. While the meal is the only practical method for outlining the *stomach*, nevertheless, it is usual to see the stomach completely outlined for a brief time only. The outline of any portion of the gastro-intestinal tract by the oral method depends upon whether sufficient barium is present at the time to fill it completely, and, consequently, this is not a wholly satisfactory method, when it is desired to obtain only the outline. The rapidity with which the barium races through most of the proximal portion of the small bowel, and the jumbling and confusion of shadows, created by well-filled loops of the terminal ileum, make it difficult or almost impossible to study the outline of the small bowel. With the colon, however, it is somewhat different and, following a barium meal, the outline in parts, may be fairly satisfactorily studied but, as has been stated, visualization depends upon the amount of barium that may remain in a segment during the period of observation. With the barium enema, the colon can usually be completely filled from cecum to rectum and, for determining the outline and position of the colon, is by far the best method.

As the stomach presents very little of interest in this study, it will not be considered. About the only portions of the small bowel, as has already been stated, that permit study of their outline, are the terminal loops of the *ileum*, but, owing to the short time barium may remain in a given loop and on account of the constantly changing shadows, a study of this portion of the gastro-intestinal tract is best approached by a consideration of the physiological side.

The normal *colon* is sacculated and smooth. The haustra are usually definite and may be more marked in certain places than in others, usually becoming less pronounced beyond the splenic flexure. Its greatest width is at the cecum and it narrows to the sigmoid, where again it widens somewhat, terminating in a broad rectal shadow. The cecal shadow is usually broad and blunt but, in a certain small number of cases, known as the "fetal type," it is somewhat tapering.

NORMAL PHYSIOLOGY OF MOVEMENT.

Following ingestion, the barium meal may fill the *stomach* completely, but this, however, is only a momentary observation, for,

in the erect posture, the barium may rapidly settle, and, almost at once, in many cases, the lower portion alone is outlined. Almost immediately after entering the stomach, the barium is seen in the first portion of the duodenum or "cap" (Cole). The types of stomachs differ considerably in regard to shape, position and time of emptying. The cow-horn or hypertonic type, more common in the hypersthenic habitus, is one extreme, while the other extreme is the low atonic type in the asthenic individual. The differences of movement and of emptying time of the different types may vary markedly. The emptying time of the stomach is of some importance when considered in connection with tuberculous ulceration of the intestinal tract. The normal stomach empties itself, on an average, in from four to six hours but, in rare instances, in even apparently normal stomachs, complete emptying occurs as late as ten hours or as early as three hours. Consequently, it is evident that too sharp a line cannot be drawn between normal and abnormal. However, it may be said that, as a rule, the stomach is empty by the sixth to the eighth hour. The first portion of the *duodenum*, functioning much like the stomach, may be visualized during the period while barium is in the stomach.

After the barium leaves the duodenum, it is broken up, separated into small masses to a marked degree, and is traced only as flocculent shadows in the *jejunum*. The flocculent shadows may persist as long as there is barium in the stomach and, possibly, for a short time afterward. A pathological change in this part of the tract, characterized by a contraction or spasm, may so retard the flow that the outline of many of the coils is visualized. Again the flow may be "normally" slow in some cases and the barium tend to accumulate in the jejunum and may even persist there for some time. This is, however, the exception, not the rule. Barium may be seen accumulating in the terminal ileum as early as the first half hour, but, in the average normal case, it usually appears between the first and third hours.

After passing through the jejunum, the rate of passage is slowed in the *ileum* and, here, the physiology of movement can be more satisfactorily studied. Some portions are continuously under observation and there are seen two main types of movement, peristalsis and segmentation. The former is characterized by an onward rushing of columns or segments of barium, which may go a considerable distance before they come to a temporary rest. With the cessation of this particular impulse, the barium may remain motionless for some time, or there may be a breaking up of the column or

segments into smaller segments, which apparently move forward and backward, after which a column may again isolate itself and be carried along by a peristaltic wave. This process may be repeated several times before the barium finally reaches the terminal ileum, where it remains until it passes into the colon. Thus, a considerable mass of barium accumulates in the terminal coils of the ileum, making impossible, many times, the identification of many of the loops.

Segmentation, the second type of movement referred to above is characterized by repeated divisions of the barium segments into other segments, these in turn merging with others in adjacent segments (see Fig. 9). This interchange repeats itself many times, having a more or less churning effect. These segments eventually empty themselves and a similar process may be observed later on in other loops. In fact, this segmentation may be in progress simultaneously in more than one area.

The *normal rate of flow* is difficult to define and, in different individuals and under certain influences, varies considerably. Consequently barium may be seen normally all over the abdominal field or, again, similar shadow complexes may indicate a pathological process. Mills states that motility bears a direct relation to habitus. The length of time that the barium may be seen in the *ileum* is dependent upon many factors, and especially upon the emptying time of the stomach, which brings up the moot question, "Is gastric delay a result of ileal stasis or can ileal stasis be due to a gastric or other reflex?" If, as Alvarez states, each segment is a law unto itself, a delay at the sphincter, when not due to disease of the sphincter, is the result of a "telephonic message" from some distal portion of the tract to "hold things up," *i. e.*, to retard the flow.

The ileum empties itself in from five to nine hours, depending upon the emptying time of the stomach. Barium, remaining in the ileum for six hours after all the barium has left the stomach, if there is a trace in the cecum, indicates an ileal stasis (Hurst).

The cecum is usually seen in from two to four hours after ingestion and, by the sixth hour, the head of the barium meal is usually in the hepatic flexure region, though in some apparently normal gastrointestinal tracts it may advance as far as the sigmoid (see Newton, p. 51). Complete evacuation of the meal usually takes place in from thirty-six to forty-eight hours.

The cecum usually remains partially or well filled from the fourth to the twenty-fourth hour and at times, longer.

The movement of the contents of the colon is quite different from

that of the small bowel. As the barium passes into the colon from the small bowel, the ascending colon is seen to fill passively upward from the cecum, and the barium column, for the most part, remains

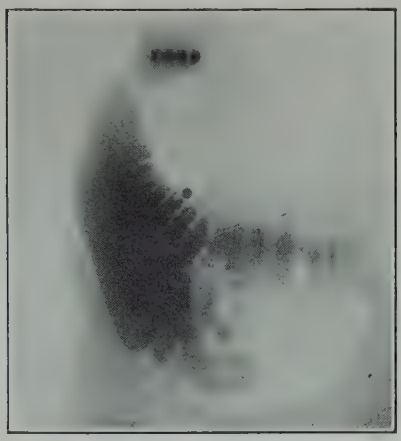

FIG. 18.—Six hours. Note the smooth haustration, also the average normal broadening of the cecum, and the position of the head of the column. Seven and eight hours confirmatory.

FIG. 19.—Twenty-four hours. Colon well filled pretty much throughout, particularly the ascending, a finding practically never seen in cases with early tuberculous ulceration.

intact until it reaches the transverse colon. After passing the hepatic flexure, an impulse may arise which isolates a mass from the main column and carries it to some distal part of the colon. This has been referred to as mass peristalsis and is probably the most

usual method of propelling the bowel-content analward. It is uncommon to see this, due probably to the fact that it occurs most frequently only after meals. The striking manifestation of mass movement is the disappearance of the haustra in that section through which the barium column is passing. When the mass comes to rest, which may be anywhere beyond its starting point, the haustra reappear. In ulcerative entero-colitis the observation of mass movement is not as uncommon as in the normal (see p. 53.)

FIG. 20.—Barium enema. Normal colon. Ileo-cecal valve competent. These were plates of a healthy individual.

Other forms of movement have been described and presumably play an important part in the physiology of movement. Mills mentions a peripheral movement and shows plates to illustrate it. It is seen as a slowly advancing thin layer of the distal portion of the impregnated mass, being somewhat delicate in outline, as the barium makes its way between the walls and the fecal matter already present and not impregnated with barium.

It is usual to find in the normal individual the cecal or proximal end of the column intact, until such time as the regular meals, taken following the barium meal, reach the proximal end of the barium column and break up its solidity. Hence, an interpretation of defective-filling at this time would have to be guarded. The reverse is also true, *i. e.*, a defective filling cannot be interpreted until sufficient barium to overfill it has passed through the suspected segment.

PATHOLOGICAL PHYSIOLOGICAL MOTILITY.

A change in motility, brought about by disease, can be manifested in two ways: (1) By hypermotility and (2) by hypomotility. In other words, a pathological process may cause an acceleration or retardation of the flow of the intestinal contents. It is not to be inferred from this that any change of motility indicates necessarily the presence of a pathological process. However, certain diseases do alter materially the rate of movement, which may be manifested either locally or generally. In tuberculous ulceration, the change in motility is usually local but may be both local and general.

It would seem reasonable to assume that the presence of an ulceration in the intestinal tract would alter the motility of the affected portion, and, in consequence, would alter the rate of flow of the intestinal contents through the affected segment, and usually throughout the rest of the tract as well. This is most often the case in ulcerative tuberculous colitis, for, whereas the normal cecum is usually well filled from the fourth to the twenty-fourth hour, (see Figs. 18 to 20), such is not the case when ulceration is present (see Figs. 25 to 26).

The hypermotility of the cecum and ascending colon depends, Keith thinks, upon the increased amount of nerve tissue about the ileo-cecal sphincter, which he terms the "ileo-colic collar." Irritation of this area, even on the ileal side of the sphincter, may produce hypermotility of the cecum and ascending colon, together with a filling-defect. While suggestive, it has been impossible for us, up to this time, to confirm this statement.

Changes in motility, as revealed by roentgen-ray study, are more dependable in the diagnosis of intestinal tuberculosis than anatomic changes. This becomes very evident when it is found that, in a great number of positive cases, the involved segment is at all times relatively or entirely free from barium. Even in many of the cases where barium enemas were administered, the diseased area refused to retain barium sufficiently long to record the outline on a film. In fact, in a certain number of cases no barium apparently entered the diseased cecum or ascending colon, and, consequently, a study of its outline was never possible. If diagnosis was postponed until such time as it might be possible to outline the diseased area, some cases would go undiagnosed for a considerable length of time.

Another point of importance, in outlining the ascending colon by enema, is that no effort on the part of the examiner can prevent the

enema from passing into the small bowel, which frequently occurs, and many times confuses the shadows of the cecum and possibly of the ascending colon. Such filling of the terminal ileum occurs often, after the injection of the barium, when it has reached the cecum, has been stopped.

Again, the presence of an ulcer, on the anterior or posterior wall of the colon, may not be seen, owing to the density of the heavy barium column, which more than compensates for the small amount of barium that may be displaced by the protruding ulcer. Lateral views might be of some assistance in these cases. However, the confusion of lateral views is obvious to those who have made use of them. The possibility of the presence in the colon of fecal matter, containing no barium and casting no shadow, is not to be overlooked when interpreting defects in outline, bearing in mind that spasm or "filling-defect" cannot be attributed to lesions in other parts of the gastro-intestinal tract. The results, noted in Table XLI, are probably the best evidence in favor of the value of pathological changes in motility in diagnosis of ulceration of the colon.

In a study of 62 cases, which presented positive roentgen-ray evidence of tuberculous ulcerative colitis, in which the diagnosis was upheld at operation or autopsy, there was noted in the many fluoroscopic and radiographic examinations made of each case, a definite localized hypermotility of the part involved, as well as, in many of the cases, a general hypermotility of the whole colon.

Exception has sometimes been taken to the use of the term "hypermotility," and other terms have been suggested to replace it, such as, hyperfunction, hyperperistalsis, etc. It matters little, what term we apply to this physiological alteration if we bear in mind the probable underlying influences that produce it. If ulceration is present, it may and probably does cause the bowel contents to move more rapidly through the affected parts, and, furthermore, it is quite conceivable that such an impulse may carry the intestinal contents considerably beyond the involved areas. Hence, it does not necessarily follow, that because the contents are seen to pass rapidly through a section beyond the point at which the impulse was started, that this area is also diseased (see Figs. 104 and 105), but it is reasonable to conclude that the origin of this hypermotility lies in the seat of trouble.

From what has been said, we can safely conclude that a state of hypersensitiveness exists in an ulcerated area, and that the passage of the intestinal contents into this portion of gut acts as an irritant, either by its contact with the ulcerated area or because it forces the

colon to dilate in order to receive it, thereby irritating the affected area, which responds in the form of a "spasm" or hyperactivity of this part. The contents are squeezed out, possibly in either direction, but as the cecum is the most common site of disease in the colon, this spasm is more likely to pass the contents analward. However, at times, it appears as though some were forced back though the ileo-cecal valve.

In support of the presence of a localized hypermotility in the diseased area, Alvarez states in his comprehensive articles on gradients in the intestinal tract, that there is a higher gradient at the diseased site and that there is an upgrade, or retardation, immediately proximal to the diseased area and a downgrade, or acceleration, immediately distal to it. This hypothesis may often explain physiologically the empty, diseased cecum, with ileal stasis often present, and the general hypermotility occurring beyond. If the peak of this altered gradient is very high, the diarrhea, so frequent in tuberculous ulceration, is partially explained, and conversely, as the ulceration heals the peak is flattened, the diarrhea is lessened and, in many instances, disappears. The process of the flattening of this peak is very often demonstrated by serial roentgenographical examinations (see Fig. 72 to 80).

The fact, that there is a retrograde flow, or *antiperistalsis*, in the proximal colon, a phenomenon that has been demonstrated by Cannon and later confirmed by Case and others, and the fact that the barium meal is constantly and rapidly hurried through the cecum or ascending colon in tuberculous colitis, emphasize further the value of interpreting correctly pathological hypermotility. Considering that we know that movement is normally more or less continuous after the meal passes the middle of the transverse colon, and that a portion of the barium column may move uninterruptedly from the hepatic flexure to the sigmoid, possibly leaving behind it a threadlike or irregular outline, simulating defective filling, the interpretation of "pathological hypermotility" or filling-defect in this region has to be *very* guarded.

In consequence of the hypertonic contraction or spasm of the ulcerated part, there is produced a marked irregularity in the outline of the barium shadow which has been referred to as a "*filling-defect.*" This isonly the result of hyperfunction of this portion of the colon and usually differs materially from the filling-defect caused by a hyperplastic type of tuberculosis or by malignant growths, where there is usually a partial obliteration of the lumen and often a very decided and permanent defect in the wall of the gut. Such con-

ditions produce the constant filling-defects, seen so frequently after the colon has been filled with barium by enema.

"Pathological motility" of the small bowel may be divided into three groups: Pathological motility (1) of the cap, or first portion of the duodenum, (2) of the jejunum, and (3) of the ileum. Few radiological observations have been made in regard to pathological motility in ulcerative conditions in the upper bowel.

The speed, with which the barium passes through the *jejunum*, renders important only one possibility in the study of a change in its motility, *i. e.*, a decided delay or stasis, but as this has not been noted in tuberculous enteritis, it is apparently of slight, if any value in diagnosis. In the *ileum*, however, where normally the movement is slower, there is more chance for study. Here again, it is a matter of attempting to determine when delay or stasis at any particular point may be looked upon with suspicion. While no definite conclusions can be drawn, stasis of a half an hour or longer, in any one coil with proximal dilatation, has been proved many times to be due to tuberculous enteritis.

In a number of cases, an *ileal stasis* of more than six hours at the sphincter has been demonstrated (see Fig. 25 and 26). When such a stasis is observed before any barium has entered the cecum, it is reasonable proof of a real stasis. However, after the ascending colon is partially filled and the stomach has been empty for some hours, the interpretation of an ileal stasis may be complicated by the phenomenon described by Case, *i. e.*, a regurgitation of the cecal contents into the ileum, which, while keeping the terminal loops of the ileum filled, may not be a true ileal stasis.

TECHNIC OF EXAMINATION.

The technic of the examination of the gastro-intestinal tract by the opaque-meal method is fundamentally very similar to that used in most roentgen-ray laboratories.

The Meal.—Certain mediums such as buttermilk, appear to alter the normal movement and, in some individuals, may cause diarrhea. However, on the whole, almost any vehicle is satisfactory, as long as it does not depart too much from what might be termed the daily diet.

The suspensions in use at the Trudeau laboratory are as follows: Malted milk, 2 ounces; barium sulphate, 4 ounces; water, sufficient to make 16 ounces.

Flour, cocoa and sugar, of each, 1 tablespoonful; barium sulphate, 4 ounces; milk, sufficient to make 16 ounces.

Preparation of the Patient for the Barium Meal.—The patient is instructed to abstain from the use of any form of laxative for thirty-six hours prior to the ingestion of the barium meal. This is advisable in order to exclude the possibility of any hypermotility, incident to its use. At a specified time, usually 8 A.M., the barium meal is taken, following which the patient eats his usual breakfast. As it has not been found necessary to observe fluoroscopically the ingestion of the barium meal in cases suspected of intestinal tuberculosis, this is usually omitted and the barium may be taken at the patient's house. However, where the esophagus or stomach is suspected of being diseased, it is obviously necessary to examine them fluoroscopically during the ingestion of the meal. Seven hours following the ingestion of the barium, the patient reports to the roentgen-ray laboratory for examination. In the interval between ingestion and the first fluoroscopic observation, the patient abstains from eating or drinking. In this way, one can estimate the emptying time of the stomach.

As an alternate technic the following may be used. The patient abstains from the use of laxatives as before. At 10 P.M., on the day previous to the examination, the patient takes two-thirds of a barium meal and the same quantity again the following morning, at 7 A.M. He eats his usual breakfast at the usual hour, then reports at the laboratory at 9 A.M. for the first observation. Following this observations are made at specified hours, which usually closely approximate those in the previously described method. This two-meal technic may present some advantages, in that portions of the tract are more continuously under observation, which is helpful when the ileum is suspected, and in that it makes possible the examination of the small bowel and colon at the same time.

In a few cases, it has been found necessary to give the meal the night before, in order that the cecum and ascending colon might be visualized the following morning, as with the usual technic it had been discovered that very little barium had passed into the colon up to the ninth hour.

The Barium Enema.—Ordinarily no preparation of the patient (*i. e.*, the use of a cleansing enema) is necessary for the administration of the barium enema, as we have found from experience that the colon can be filled completely in practically all instances. The authors feel that the morning is the best time for the administration of enemas, for the reason that the cecum is normally practically

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empty until about four hours after breakfast, which observation has also been noted by Hurst. Consequently, this portion of the colon is ready to receive the injection without having to resort to irritating laxatives and soap-suds enemas. When the occasion arises, a mild laxative may be used the night before, in order to free the colon from anything that might possibly interfere with the administration of the barium enema. As this interference is usually in the sigmoid and rectum, it is rarely necessary to use it.

The Roentgen-ray Examination.—It is necessary that the eyes of the examiner be fully accommodated to the dark, before the screen examination is started, as slight changes may otherwise be overlooked. The length of time for complete accommodation varies according to the brightness of the day. Under the average conditions ten minutes are sufficient. Protection against the rays is of importance particularly to the examiner, and considerable care should be exercised in this respect.

Seven hours following the ingestion of the barium meal, the patient reports to the roentgen-ray laboratory for fluoroscopic and radiographic examination. From this time on, observations are made at one-half-hour or three-quarter-hour intervals until the ninth or tenth hours following the ingestion. The necessity for the repeated observations depends directly upon what may have been noted at the previous examination, *e. g.*, a doubtful finding would increase the number of observations, whereas a frankly positive or negative finding would reduce their number.

Considerable emphasis should be placed upon this period of observation, for it is at this time that a diagnosis is possible in a great majority of cases. Usually there is enough barium at this time both in the ileum and transverse colon to permit the study of the movement of the intestinal contents, and, as the diagnosis of a diseased bowel is made primarily upon a marked alteration in the rapidity of movement, it can readily be seen that close study is at this time necessary, and so the necessity for repeated observations is emphasized. At the observation at the seventh hour, the ulcerated cecum may be partially filled with barium, whereas at the next observation, one-half hour later, all the barium may have been eliminated, due to a spasm of this portion of the colon. Again at the eighth hour, more barium is found in the ulcerated segment, only to be ejected a little later. The value of repeated observations is seen when one is dealing with pseudo-filling-defects. At one observation two or three sacculi are free from barium (see Figs. 43 to 48, 49 to 51) and a filling-defect might be interpreted, but

would probably be wrong, for, at a later examination, this portion of the colon was well filled at all observations. It is a question, as to whether an ulcer in a sacculus would cause only that sacculus to contract. In all proved cases, the filling-defect involved the whole segment. Then again, the filling-defect may possibly extend beyond the diseased area. To be of diagnostic value a filling-defect should be present at a majority of observations, and, when appearing only once, would have to be confirmed at a later examination.

It must never be overlooked that, from a study of the plates alone, it may be impossible to separate the shadows cast by the ileum from those of the cecum and ascending colon. Only a fluoroscopic examination or an enema will in these cases render the plate intelligible, but, in a study of many plates, taken in the laboratory and at the bedside, this confusion has rarely arisen. The necessity for such differentiation depends upon the fact that a loop of the ileum may underlie the cecum and ascending colon and suggest a filling-defect.

During the period of observation, which is usually from 3 P.M. to 6 P.M., the patient remains at or near the laboratory, subject to call from the examiners. The patients are permitted to eat after the first observation. In fact, this is recommended, for the reason that it helps to carry out the daily dietary régime, and also serves the purpose of inducing, during the period of observation, peristaltic movement, which very frequently can be observed fluoroscopically. Following this series of observations, the patient is again examined the following morning, about twenty-five hours after ingestion. This usually completes the observation of the barium meal.

A day or two later, a barium enema may be administered. This is done with a gravity pressure as low as possible, usually 12 to 18 inches, with the patient in the prone position. The injection is controlled by fluoroscopic examination and should be stopped, if the patient's discomfort is too great. After having filled the colon, a film is made in the prone or supine position. Quite frequently before the cecum fills completely, the enema passes into the ileum, which should be avoided as much as possible, as the barium-filled coils of the ileum often obscure the outline of the ascending colon.

Examination by the Two-meal Technic.—Two hours following the ingestion of the second meal, and obviously eleven hours after the first meal (see p. 183), the patient reports to the laboratory for the first observation. As the small bowel is to be studied especially at this time, the observations should be made at short intervals of about twenty minutes each, and continued for one and a half

hours, depending upon what is seen at each observation. Following these observations the patient is again observed six hours later. This would be about eight hours after the second and nineteen hours after the first meal. From this time on, the observations and handling of the case are the same as for the one-meal technic.

ROENTGENOLOGICAL DIAGNOSIS OF INTESTINAL TUBERCULOSIS.

A brief reference to the pathological anatomy, upon which the roentgenological interpretation is based, may be advisable at this time (see p. 60). Following the development of the tubercle, degeneration and caseation occur and finally ulceration. This process undoubtedly alters the movement of the affected portion of the tract and usually manifests itself by a hyperactivity, which, in turn, produces a partial or complete elimination of the contents of the affected portion. If this abnormal impulse or hyperactivity is strong enough, it may do more than empty just the affected portion, and may even propel all the bowel contents, distal to it, into the sigmoid or rectum. Such overactivity, or hyperfunction, is the ground-work of the roentgenological diagnosis of tuberculous ulcerative colitis. On the fluoroscopic screen or roentgenogram this hypermotility is characterized by "filling-defects" and frequently a generalized hypermotility.

As the authors have previously stated, the first rather marked variation from the normal, that they noted in cases suffering from ulcerative colitis, was that the bowel emptied itself in a much shorter time than occurred in the normal individual. The barium was completely eliminated in less than twenty-four hours, and in some cases as soon as fifteen hours. In one case it was all in the sigmoid and rectum in twelve hours (see Figs. 54 and 55). As no laxative had been used previous to the examination, obviously, this hyperactivity needed explanation. Upon closer observation it was found that the origin of this *generalized hypermotility* was in the ileo-cecal region. Whereas the cecum was normally filled from the fourth to the twelfth hours, it was found to be constantly empty in some of the cases (usually the more advanced) and only partially filled in others. A typical illustration is seen in Figs. 25 and 26. At no observation on this case was the proximal colon visualized. Only faint traces of barium were seen in the ascending colon in the earlier observations and, as the ileum was still emptying, it was reasonable to conclude that the barium was passing rapidly through the colon.

At the twenty-fourth hour, practically all the barium had been eliminated. It was evident, then, that there must be a site of irritability somewhere in the proximal colon. This type of extreme irritability, or hyperactivity, is usually present in the more advanced

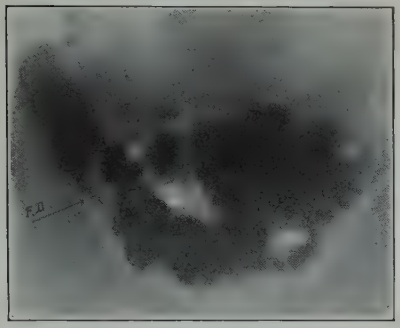

FIG. 21 (Case 157) seven hours.—Roentgen finding: filling-defect (*F.D.*) at the cecum; generalized hypermotility (head of the column in the sigmoid).

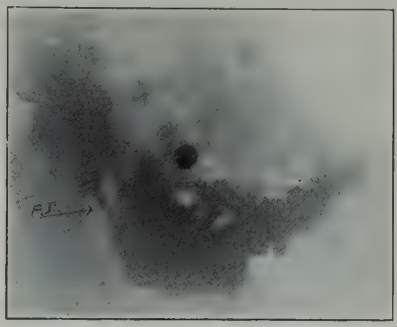

FIG. 22 (Case 157) eight hours.—Roentgen finding: filling-defect (*F.D.*) still persistent.

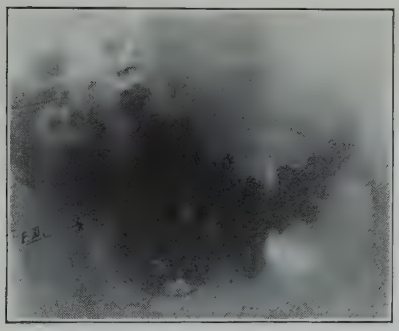

FIG. 23 (Case 157) nine hours.—Roentgen finding: persistence of filling-defect (*F.D.*) of the cecum.

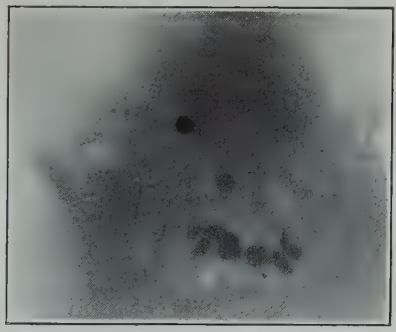

FIG. 24. (Case 157) twenty-five hours.—Roentgen finding: generalized hypermotility, barium almost completely eliminated.

Clinical Finding.—Advanced pulmonary tuberculosis (cavities right and left). Artificial pneumothorax. Fistula-in-ano. Roentgen-ray and clinical evidence of colitis. Ultraviolet-ray treatment since June, 1923. Occasional elevation of temperature accompanied by vomiting, nausea, abdominal pain, "biliousness," but no diarrhea. Tuberculous laryngitis, 1922. Later, diarrhea and cramps. Fair results from pneumothorax. Died, January 7, 1925.

cases. However, cases are often found in apparently early intestinal ulceration, that reveal a decidedly generalized hypermotility (see Figs. 27 and 28). Where this hypermotility is not so pronounced and is more localized, the ulcerated area (usually in the cecum) tends to retain barium for a longer period of time, though it is rarely

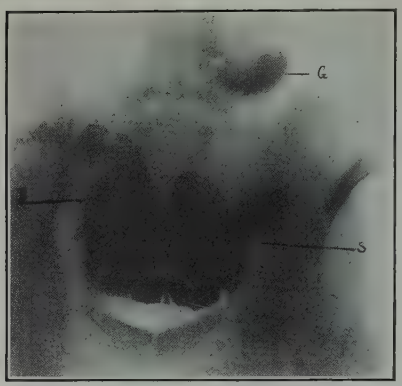

FIG. 25 (Case 7875).—Roentgen finding at six hours: gastric retention (*G*); persistent segmentation and marked dilatation of the terminal loops of the ileum (*D*); hypermotility (head of the column in the sigmoid "*S*"); the ascending, transverse and descending colon practically empty. Confirmed at seven hours.

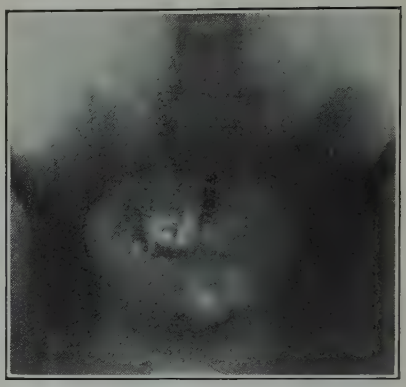

FIG. 26 (Case 7875).—Roentgen finding at twenty-four hours; generalized hypermotility; barium almost completely eliminated.

Clinical Finding.—Female , single, aged twenty-two years. Advanced pulmonary tuberculosis of whole right lung, with cavity at apex, began nine months previously. Pain, meteorism, loose bowels, two to five a day. Emaciated, weak. Died six months later. No special treatment.

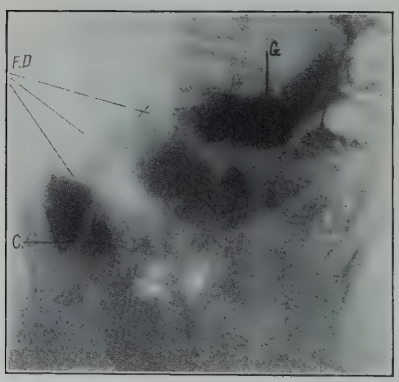

FIG. 27 (Case 110).—Roentgen finding at six hours; marked gastric retention (*G*)—note the absence of barium (*F.D.*) in the hepatic flexure region, confirmed at subsequent examinations; filling-defect "*C*" crossing the cecum (but no localized hypermotility); generalized hypermotility (head of the column in the sigmoid).

FIG. 28 (Case 110).—Roentgen finding at twenty-four hours: generalized hypermotility, all the barium having been eliminated. Patient constipated.

Operative Finding.—See Case 110. Table XXXVI.

Clinical Finding.—Male, married, aged thirty-three years. Began with "stomach trouble," December, 1917. Pulmonary tuberculosis diagnosed. In March, 1918 infiltration upper halves right and left lungs. Bowels loose at times and markedly constipated at other times. Operation; excision followed by death in one week.

filled to its capacity. This state of affairs has given rise to the term "*filling-defect.*" The characteristic "filling-defect" is seen in Figs. 29 to 31 and resembles very much the result that might be produced, if the cecum, filled with barium, was squeezed by the hand. In reality the cecum is, or has been, in a state of spasm and

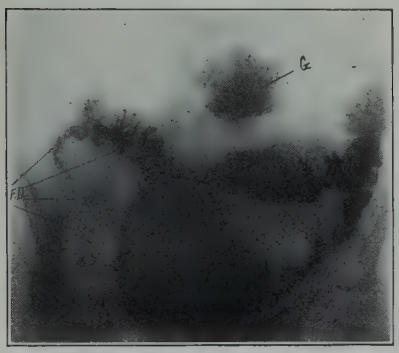

FIG. 29 (Case 143).—Roentgen finding at six hours: gastric retention (*G*); filling-defect or spasm of the colon from the cecum to the hepatic flexure region (*F.D.*), generalized hypermotility (head of the column in the sigmoid).

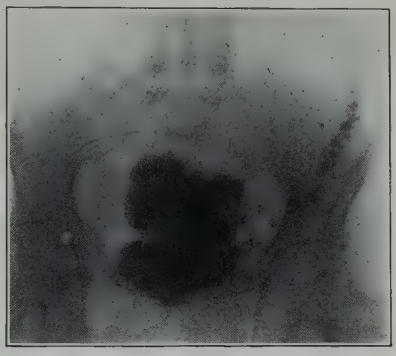

FIG. 30 (Case 143).—Roentgen finding at twenty-four hours: generalized hypermotility.

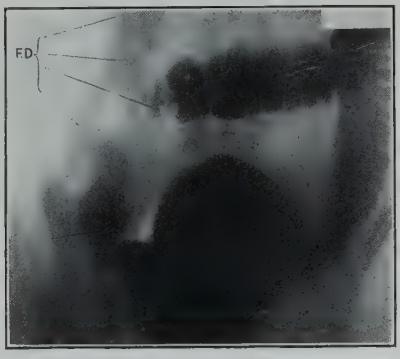

FIG. 31 (Case 143).—Roentgen finding after barium enema: filling-defect and spasm of the ascending colon and hepatic flexure region (*F.D.*) confirming the examination by meal.

Operative Finding and Roentgen Interpretation.—See Case 143. Table XXXVI.

Clinical Finding.—Male, single, aged twenty-six years. Pulmonary tuberculosis probably began in 1915. In March, 1918, infiltration of right lung and left upper one-half. Abdominal symptoms, considerable flatulence, discomfort. Examination showed movable thickening in the region of the cecum. Tuberculous colitis diagnosed, May, 1918. Operation, June, 1918. December, 1918, artificial pneumothorax right side.

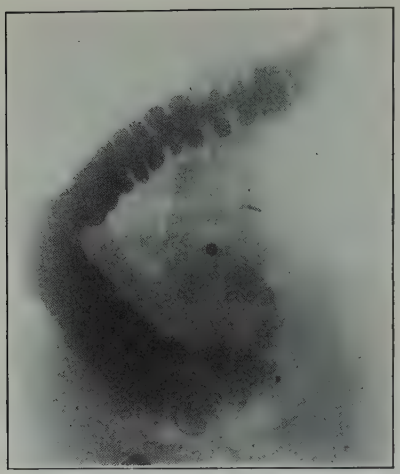

FIG. 32 (Case 112).—Roentgen finding at seven hours: negative. Confirmed at eight and twenty-four hours.

FIG. 33 (Case 112).—Thirteen months after first examination. Roentgen finding at seven hours: filling-defect or spasm (*F.D.*) of the cecum. Confirmed at eight hours.

FIG. 34 (Case 112).—Roentgen finding at nine hours: note that the cecum is slightly better filled than at seven hours; filling-defect (*F.D.*) still very definite. At twenty-four hours generalized hypermotility, all barium having been eliminated.

Autopsy Finding.—See Case 112. Table XXXVI.

the barium remains only, where it has found a lodging place, in the folds of the contracted cecum.

When the positive cases are repeatedly fluoroscoped from the seventh to the tenth hour, the cecum often appears as though it were going to fill completely, but, apparently, when the tension in the ulcerated area reaches a certain point, the irritation becomes too great and a "pathological spasm" occurs, in an attempt to free the diseased area of its irritating contents (see Figs. 32 to 34).

Barium is very frequently seen to pass from the ileum into the cecum or ascending colon. In cases, where there is no ulceration of this area, there is no immediate effort on the part of the colon to rid itself of the barium, and the bowel is filled and may be distended. It is worthy to note at this time that a well-filled ascending colon, in which there is no ulceration, can be palpated and, in fact, a manual effort made to initiate a peristaltic wave, but usually to no avail. The colon shows no tendency to pass the barium analward and hence the ascending colon remains filled.

However, in cases where ulceration has later been proved to exist, there is often seen a perceptible movement on the part of the cecum, apparently set in operation by the entrance of the ileal contents. The cecum may or may not contract with each squirt of barium that enters it, but as the cecum fills, and in consequence is somewhat distended, and also as more material comes in contact with the ulcerated or inflamed area, the diseased portion may, and usually does, respond with a contraction in the nature of a spasm. It is not uncommon to see peristaltic mass movement in cases of ulcerative colitis, which is in marked contrast to what is seen in normal individuals, where it is seldom observed. Confirmation of this observation has been noted by Case, who declares that the contents of the colon can be shifted little, if any, by palpation, and who quotes Schwartz as saying "that even with strong pressure it is not possible to lift the content of the ascending colon into the transverse."

The mechanism in ulcerative colitis is, however, much the same as that described for the normal cases: a contraction is seen to isolate a segment or mass from the main column, whatever haustra are present disappear, and the mass for a few seconds moves forward, apparently more rapidly than in the normal cases. When it comes to rest, the haustra may reappear. This, however, does not always happen, due probably to the fact that there is ulceration in this region also. From this, it is not to be inferred that if the haustra appear there is no ulceration, for, in proved ulcerative cases, haustra

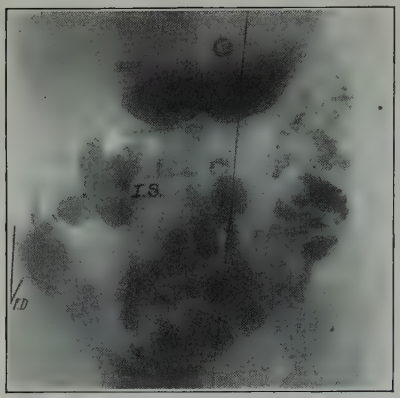

FIG. 35 (Case 127).—Roentgen finding at six hours: gastric retention; persistent segmentation of small bowel (*I.S.*); filling-defect (*F.D.*) of the ascending colon. Confirmed at seven hours.

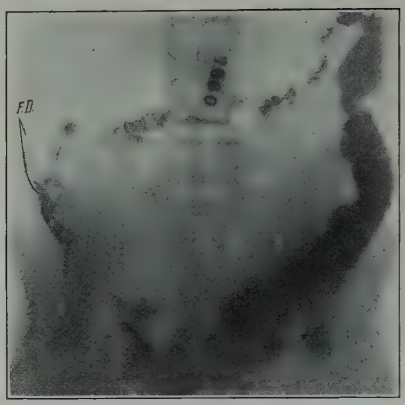

FIG. 36 (Case 127).—Roentgen finding at twenty-four hours: uncommon residual in the proximal colon (*F.D.*) suggesting barium remaining in the craters of the ulcers. (Compare with Fig. 71.)

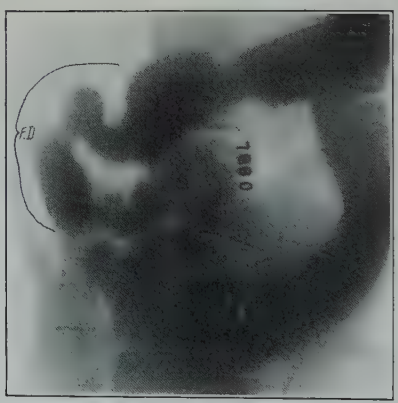

FIG. 37 (Case 127).—Roentgen finding after barium enema: lack of haustration fairly well throughout, more marked in the proximal colon; filling-defect (*F.D.*) of the proximal colon; incompetent ileo-cecal valve, barium having passed into the ileum.

Operative Finding.—See Case 127. Table XXXVI.

Clinical Finding.—Male, married, aged thirty-seven years. Pulmonary tuberculosis began January, 1917. In June, 1918 infiltration upper two-thirds of right and small portion of left lung. Intestinal symptoms began September, 1917 with occasional diarrhea. Diagnosed, July, 1918. Operation, short circuiting, pain and discomfort much increased. Died, October 19, 1918.

have been present in the diseased area. This condition, however, is usually seen with ulceration in the distal colon, rarely in the proximal.

At times during the earlier observations, from seven to ten hours, the ulcerated area (usually in the ascending colon) may be fairly well filled, though the outline is ragged. With moderate palpation, however, a stimulus may be started and the barium is seen to move forward to some distal portion of the colon. This phenomenon is often demonstrated in positive cases, but occurs very rarely in the normal colon. From this observation, it would again seem probable that the added irritation (increased internal pressure), brought about by palpation, was all that was needed to start a peristaltic rush.

In the small bowel the same altered movement is apparently present, that is, at the site of the ulcer, there is usually a spasm or hyperactivity, but, in view of the fact that there is normally a more rapid flow in the small bowel, the abnormal hypermotility passes unnoticed. In fact, quite the reverse is noted for, as a result of the spasm, there is a temporary stasis of the barium just proximal to it. This delay is often present for a considerable period of time, and, in an effort to overcome this obstruction, a temporary dilatation of the proximal segment develops (see Figs. 35 to 37). This condition has given rise to the term, pronounced or persistent segmentation and dilatation, which changes are the only manifestations upon which a diagnosis of enteritis may be based. The longer the delay persists in a segment, obviously the greater the probability that a lesion exists. It is often wise to repeat the examination to determine whether the segmentation and dilatation recur in the same loop or loops of the small bowel.

FILLING–DEFECTS.

It is a question, whether the term, filling-defect, is not a misnomer when used in the interpretation of tuberculous ulcerative colitis. The word implies a defect, usually permanent, in the normal outline of the barium column, and, in turn, suggests a permanent alteration in the wall of the organ, containing the barium, such as that brought about by a hyperplastic tuberculosis, a gastric ulcer, a chronic ulcer of the duodenum, an annular cicatrix or some forms of malignant growth involving the gastro-intestinal wall or encroaching upon it from without. Adhesions, as well as chronic deep tuberculous ulcers, may also produce a permanent alteration in the outline of the the normal barium shadow. These, however, differ

essentially from the filling-defects, seen in early tuberculous ulcerative colitis. The irritation of even a recent small ulcer on the mucosa of the cecum, not sufficiently deep to cause a defect in the outline of the barium column, may bring about spasm of the cecum, producing a "filling-defect" of the whole cecum, or ascending colon, whereas the actual defect in the wall may be only 5 mm. in diameter. So, while a defective filling of the whole cecum or ascending colon in tuberculous ulcerative colitis may be due to a slight lesion, a similar filling-defect, when caused by a malignant growth or by

Fig. 38 (Case 5384).—Roentgen finding at seven hours: negative for ulcerative colitis. Confirmed at eight, nine and twenty-four hours.

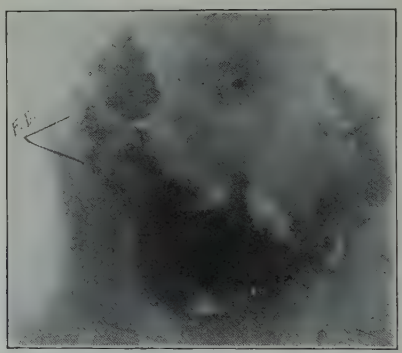

Fig. 39 (Case 5384).—Nine months after first examination. Roentgen finding at seven hours: filling-defect or spasm (*F.D.*) of the ascending colon. Confirmed at eight and nine hours. Roentgen finding at twenty-four hours: generalized hypermotility, all the barium having been eliminated.

Clinical Finding.—Female, single, aged seventeen years. Pulmonary tuberculosis began October, 1919. In June, 1920, infiltration left upper two-thirds. At this time no evidence of tuberculous colitis. Later, increase of infiltration in right and left lungs; cavities. Later, developed abdominal symptoms. Tuberculous colitis diagnosed, November, 1920. Died, 1921.

hyperplastic tuberculosis of the colon, may be due to a permanent, and possibly an almost complete, obliteration of the lumen. A filling-defect in tuberculous ulcerative colitis may change materially from hour to hour, while the other usually remains relatively the same at each observation. This observation is further verified by the use of the barium enema, for, whereas, after the barium meal, we see a filling-defect or more properly a pathological hypermotility or spasm of the cecum, when the enema is administered, the outline may be perfectly normal. Such is not the case where the wall of the

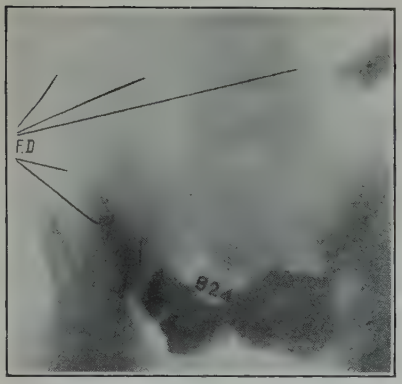

FIG. 40 (Case 136).—Roentgen finding at six hours: marked filling-defect and spasm of the proximal colon (*F.D.*) with questionable filling-defect distal to this site; generalized hypermotility (head of the column in the sigmoid). Confirmed at seven hours.

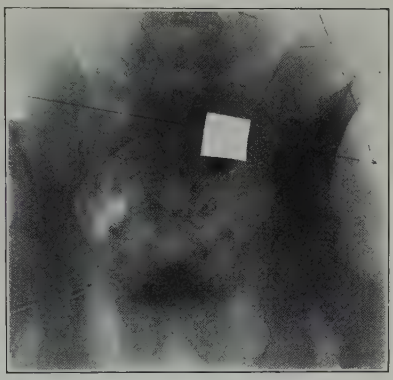

FIG. 41 (Case 136).—Roentgen finding at twenty-four hours: generalized hypermotility, barium almost completely eliminated.

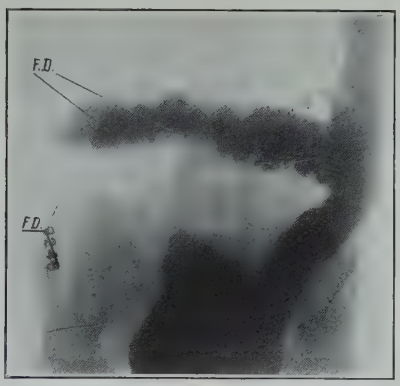

FIG. 42 (Case 136).—Roentgen finding after barium enema: filling-defect and spasm of the ascending colon (*F.D.*) and filling-defect of the first portion of the transverse colon, confirming examination by meal.

Operative Finding.—See Case 136. Table XXXVI.

Clinical Finding.—Male, single, aged twenty-five years. Moderately advanced pulmonary tuberculosis. Some diarrhea while in army in France. Short circuiting operation of some help. Later colostomy opening and irrigation of much benefit. Ultraviolet-ray treatment boosted patient as well as roentgen-ray treatment. Death followed operation to close artificial anus.

gut is involved in a malignant growth. Here, we have usually a gross alteration, producing a permanent and constant defect in the outline of the barium column, which is usually a localized manifestation. However, spasm of this segment may occur also when ulceration develops, as in early ulcerative tuberculosis. It is quite probable that chronic, deep tuberculous ulcers, with connective tissue and proliferative changes, will manifest themselves in much the same way as malignant tumors, by producing a real defective filling.

PSEUDO-FILLING-DEFECTS.

A pseudo-filling-defect naturally implies that type of irregularity, which, in the estimation of the interpreter, is not wholly characteristic and indicates there is no pathological change underlying it. Owing to the multiplicity of irregularities that do occur in the filling

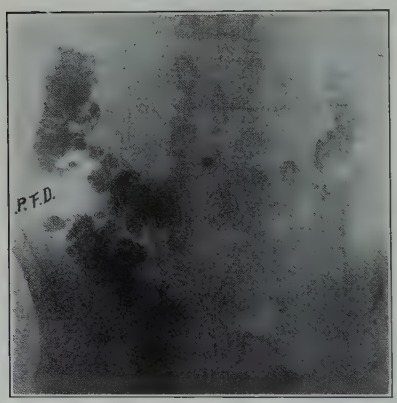

FIG. 43 (Case 6638).—Roentgen finding at seven hours: absence of barium in the middle of the ascending colon, pseudo-filling-defect (*P.F.D.*). A gas bubble at this site probably the cause for the lack of barium. This area was filled at the eighth-hour observation (see Fig. 44). Note there is no spasm at this site.

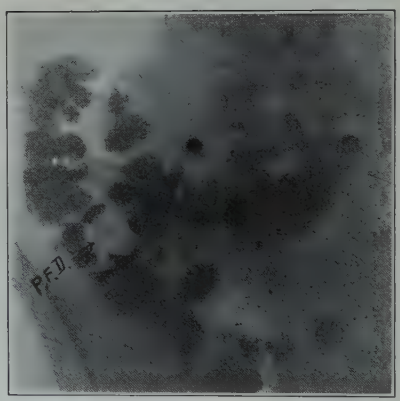

FIG. 44 (Case 6638).—Roentgen finding at eight hours: the portion (segment) of colon previously empty (see Fig. 43) now well filled with fair haustra; note a new area of pseudo-filling-defect (*P.F.D.*). Same finding at nine hours.

of any portion of the gastro-intestinal tract, a hard-and-fast rule for pseudo-filling-defects cannot be established. However, from experience one acquires a certain degree of analytical ability that enables him to disregard certain irregularities in outline, *especially when on further observation the picture is negative.* It is quite obvious that small amounts of barium entering the ascending colon are insufficient to fill it, and, in consequence, the true outline of this

FIG. 45 (Case 6638).—Roentgen finding at twenty-five hours: average normal residual.

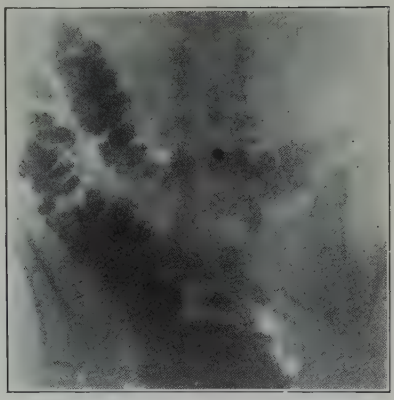

FIG. 46 (Case 6638).—Second examination five days later to confirm pseudo-filling-defect. Roentgen finding at seven hours: average normal outline and motility.

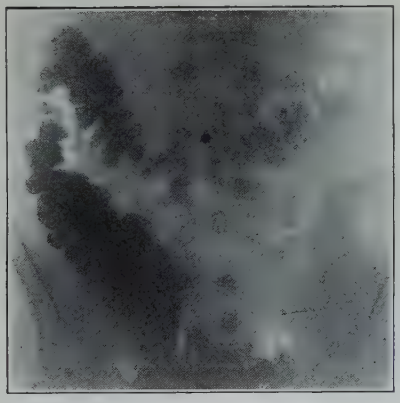

FIG. 47 (Case 6638, second examination).—Roentgen finding at eight hours: haustra smooth and regular; average normal motility; confirming seven-hour observation; colon apparently normal contradicting pseudo-filling-defect at first examination.

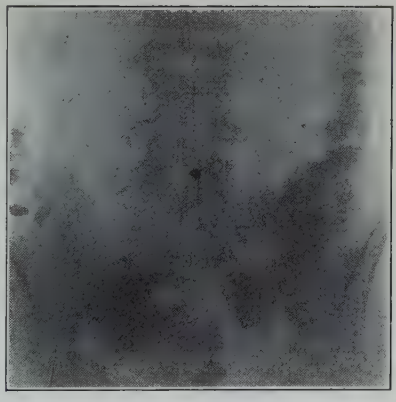

FIG. 48 (Case 6638, second examination).—Roentgen finding at twenty-five hours: average normal residual confirming previous examination.

Clinical Finding.—Male, single, aged twenty-eight years. Pulmonary tuberculosis began September, 1924. December, 1924, infiltration right upper half, left apex. No abdominal symptoms. Clinically no suspicion of intestinal ulceration. Present condition good.

portion of the colon is not seen. It is also possible that sufficient barium to fill it may not do so completely, due possibly to certain but few factors.

For instance, it is a fact that there may be continual movement of the colon at all times, not sufficient to be observed on the screen, but gross enough to affect the smooth outline of the haustra on the plate. These slight "muscular contractions" might obviously cause an irregularity in otherwise smooth haustral sacculations. This seems highly probable, particularly in very nervous individuals, where one sees what might be termed muscular irritability, a state often referred to as spastic colon.

Another factor of importance is the character of shadow which may remain, following a normal peristaltic wave or mass movement. Such a contraction may not necessarily carry every vestige of barium before it; therefore, the irregular stringy effect, left behind, cannot be interpreted as a filling-defect, in the sense that it indicates a pathological condition. However, these peristaltic waves, or mass-movements, are commonly seen at the distal end of the barium column, and differ, in location, from the "pathological spasm" or contraction in ulcerative tuberculous colitis, which is seen usually near the proximal end of the barium meal. Here again, one can see the danger of interpreting filling-defects beyond the hepatic flexure.

Another cause of pseudo-filling-defect may be that fecal matter, not impregnated with barium, can be present in the barium-filled colon, in consequence of which, the outline is ragged in places. Succeeding observations usually determine whether one is dealing with true filling-defects or spasm, or pseudo-filling-defects, brought about by the two foregoing factors.

"Saddling" of the transverse colon across the spine or pressure by any extrinsic force, may bring about an irregularity that may be very puzzling. This pressure may be of such a character as to keep a segment of bowel continually empty.

Repeated observations and palpation, during fluoroscopic examination, is of extreme value in ruling out pseudo-filling-defects, but, nevertheless, there are times when one is still in doubt. Another examination at a future time, for instance in a couple of weeks, is often very helpful.

The lack of pliability as described by some writers, or a local contraction of a single sacculus as described by Elliott and Cannon, and mentioned by Hurst, may definitely affect the smooth regular outline and cause confusion. The latter is more apt to happen fol-

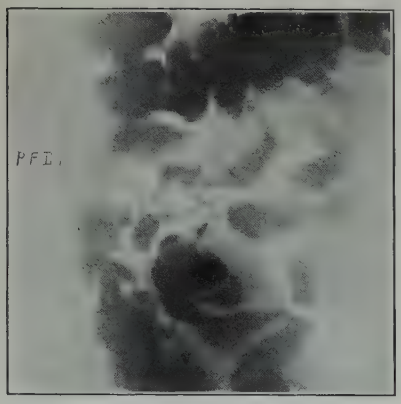

FIG. 49 (Case 6416).—Roentgen finding at seven hours: pseudo-filling-defect in the upper portion of the ascending colon (*P.F.D.*). See Figs. 50 and 51.

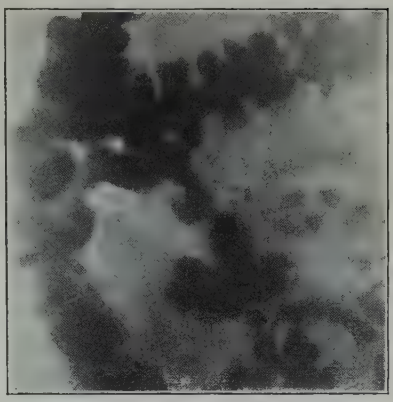

FIG. 50 (Case 6416).—Roentgen finding at eight hours: disappearance of pseudo-filling-defect; ascending colon now fairly well filled with fairly good haustration.

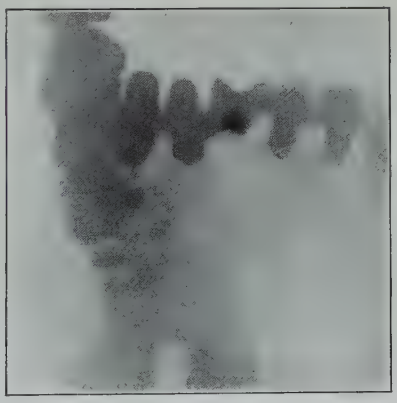

FIG. 51 (Case 6416).—Roentgen finding at twenty-four hours: well filled ascending and proximal colon. It is extremely rare to find an ulcerated area retain barium up to the twenty-fourth hour: The patient had no symptoms at any time and has remained well since. The examination was a routine one.

Clinical Finding.—Female, single, aged twenty-six years. Pulmonary tuberculosis began January, 1919. In February, 1924, slight infiltration left to clavicle. Routine examination: bowels irregular; constipated. Patient has remained well since.

lowing a meal. Here the enema may help to clear up the doubt. Examples of this can be seen in Figs. 43 to 48. It is to be noted that, in the subsequent observations of Case 6638, the previous pseudo-filling-defect has disappeared and that the sacculi are well rounded.

Other influences, that may bring about irregularities, are sharp angulations at a flexure, pressure of one segment of the colon against another, in fact, pressure, pulling (as from adhesions), or tensions of any character.

A comparison of the figures in Case 24, page 202, with those Case of 157, page 187, shows the difference between real and pseudo-filling-defect, as the diagnoses in both cases were proved at operation.

THE ENEMA.

Obviously, the object of a barium enema is to outline the colon and, possibly, to reveal some phase of movement. At the outset, it must be borne in mind that a barium enema produces, so to speak,

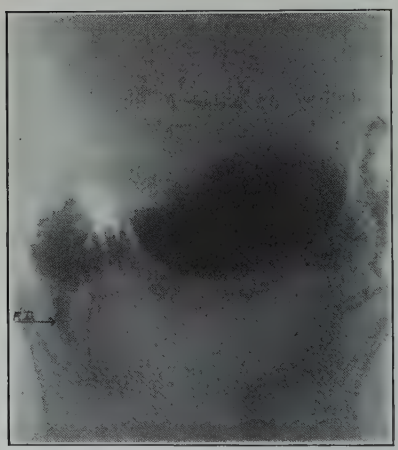

Fig. 52 (Case 133).—Failure of enema. Roentgen finding at six hours: gastric retention; the stomach is low; general hypermotility (head of the column in the rectum); filling-defect or spasm (*F.D.*) of the cecum.

Fig. 53 (Case 133).—Roentgen finding following barium enema: tapered cecum (persistent fetal type?); incompetent ileo-cecal valve, barium having passed into the ileum; note the absence of filling-defect or spasm which was observed at six hours following barium meal. Examination by enema negative.

Operative Finding.—See Case 133. Table XXXVI.

an unnatural condition in the colon. We seek to detect a defect in the outline of the colon by its use, but, as we have already stated, a spasm can simulate a filling-defect and hence, we might be led to

interpret a spasm of the colon in terms of a pathological change. But this should not be done, for a spasm, or a physiological contraction of the sigmoid or descending colon frequently occurs in apparently normal colons. However, a segment of the colon, which cannot be filled at any time (a filling-defect), is in an entirely different state from one which fills and contracts, or "spasms." Again, a segment, that will not fill, may be in tonic contraction and an interpretation of this, as due to disease at this site would be unwar-

FIG. 54 (Case 112).—Failure of enema. Roentgen finding at twelve hours: generalized hypermotility; practically all the barium is in the sigmoid and the rectum, a small amount still remaining in the terminal ileum; note the colon is empty as far as the sigmoid.

FIG. 55 (Case 112).—Roentgen finding following barium enema: colon is filled throughout; no spasm or filling-defects; incompetent ileo-cecal valve, barium having passed into the ileum; note that the enema failed to confirm the finding by meal.

Operative Finding.—See Case 112. Table XXXVI.

Clinical Finding.—Female, married, aged twenty-one years. Began with pulmonary tuberculosis in the spring 1918 while pregnant. Worse after puerperium, October, 1918. Cavities here and there throughout right lung. Intestinal tuberculosis, October, 1918. Exploratory laparotomy. Died, April 12, 1919, of pulmonary tuberculosis.

ranted, as, for example, in some cases of chronic appendicitis, where spasm of other portions of the tract may occur as a reflex. Consequently, contraction or spasm of distal segments of the colon are of very little value, but, if confirmed by a barium meal, may suggest a correct diagnosis. Spasm of the cecum, however, is of more value. After having filled the colon throughout, one does not expect to see a contraction or spasm localized to the cecum and, when, as is observed in tuberculous colitis, the cecum or ascending colon repeatedly fills and empties, this finding is of extreme importance,

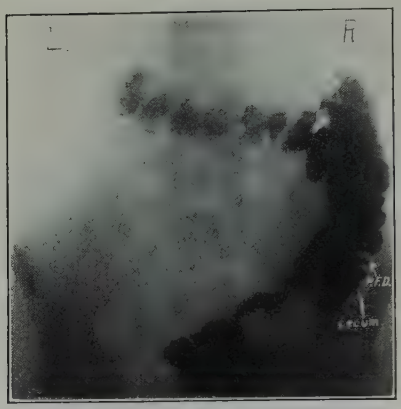

FIG. 56 (Case 24).—Failure of enema. Roentgen finding at seven hours: negative. Confirmed at eight hours.

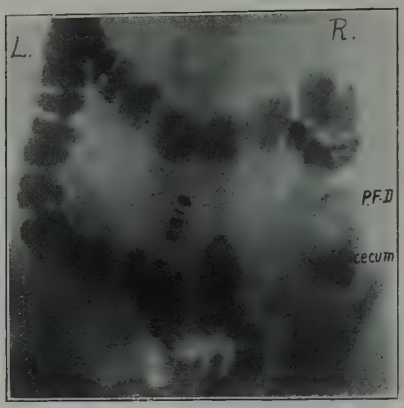

FIG. 57 (Case 24).—Roentgen finding at twenty-four hours: colon fairly well filled throughout; the retention in the cecum is normal, confirming the earlier observations.

FIG. 58 (Case 24).—Roentgen finding after enema: colon well filled throughout with the exception of the ascending portion where there is a spasm and filling-defect. At operation no ulceration was found, confirming the meal but contradicting the enema.

Operative Finding.—See Case 24. Table XXXVII.

Clinical Finding.—Female, married, aged twenty-five years. Pulmonary tuberculosis began May, 1918. Infiltration right and left upper lobes, August, 1918. Abdominal symptoms, considerable pain, nausea, tenderness right lower quadrant. In September, 1918, operation for appendicitis. Chronic non-tuberculous adherent appendix removed.

particularly if this area has been suspected during the study of the barium meal. During the administration of the enema, the diseased area appears often to be in a state of spasm, and the enema is seen to work its way slowly into the contracted segment, casting thready shadows (see Fig. 86). This spasm may, however, relax and the cecum fill partially, only to contract and empty again, which may be repeated several times. Very often this contraction or spasm extends, well beyond the supposedly diseased area. In 1 case (see Figs. 103 and 104, there was an expulsion of all the barium from the cecum

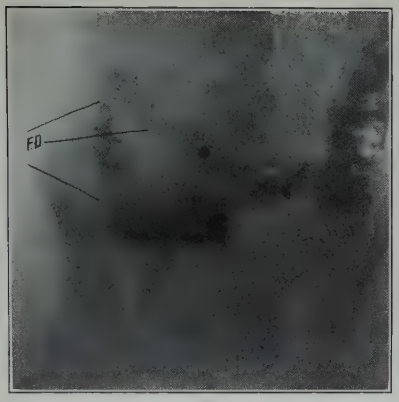

FIG. 59 (Case 146).—Roentgen finding at six hours: generalized hypermotility (head of the column in the rectum); filling-defect (*F.D.*) and spasm of the ascending colon and the first half of the transverse colon. At twenty-four hours generalized hypermotility, all the barium having been eliminated.

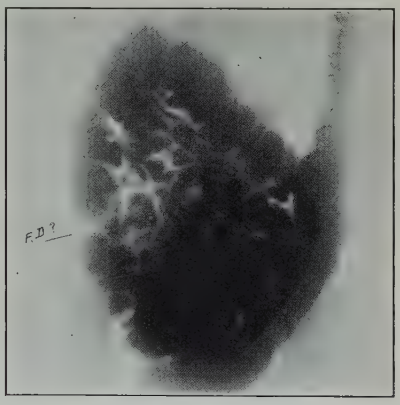

FIG. 60 (Case 146).—Roentgen finding after barium enema: probable filling-defect (*F.D.?*) of the cecum; incompetent ileo-cecal valve; note that the enema fails to reveal the extent of the lesion shown by the meal. See operative finding.

Operative Finding.—See Case 146. Table XXXVI.

Clinical Finding.—Female, single, aged seventeen years. Pulmonary tuberculosis began two and a half years previously. Infiltration of most of right and upper one-fourth of left lung. Intestinal tuberculosis diagnosed, December, 1918. Excision of bowel, April, 1919. Died, May 21, 1919.

to the splenic flexure, and it seemed very unlikely in this case that all this area was involved. The reverse is also true, *i. e.*, a well-filled colon does not exclude ulceration, for in a number of cases, proved to have ulceration, the enema completely filled the colon and there was no filling-defect or spasm to be seen (see Figs. 52 and 53).

Summary.—As has been previously stated, a pathological process in the intestinal tract will probably change its movement or outline, and, in practically all cases, a differential interpretation will have to be based mainly on variations occurring in the movement or

outline. It will be seen at once, that such an interpretation must be very inexact in many cases, for the reason that a hyperplastic type of tuberculosis of the cecum can produce the same degree of delay as any other obstructive process. However, in the case of ulcerative colitis, such a delay is extremely uncommon, whereas, in cases of tuberculous enteritis, it is more frequently seen. Summarizing, we can then say that certain variations in the outline or movement of the intestinal tract may suggest certain pathological processes, and may help materially in arriving at an ultimate diagnosis, when combined with the clinical evidence. This is well exemplified in all cases that have definite pulmonary tuberculosis, where the ulceration in the bowel is more likely to be tuberculous.

CHAPTER XXIII.

DIAGNOSIS OF INTESTINAL TUBERCULOSIS ROENTGEN-RAY METHODS (*Continued*).

DATA UPON WHICH THE ROENTGEN-RAY METHOD OF DIAGNOSIS IS BASED.

Since 1918, 2595 patients have been studied in the roentgen-ray department at Trudeau, in regard to the presence or absence of intestinal tuberculosis. In all these patients, a diagnosis of pulmonary tuberculosis had caused them to seek treatment. In 869 patients, a positive diagnosis was made and, in 1726, tuberculous colitis was excluded. Of the negative group, 46 went to operation or autopsy and in 44 tuberculous colitis was absent, while in 2, it was present. These 2 cases are of interest. In 1, a tuberculous appendix was present and the ulceration in the appendix extended over on the wall of the cecum such a short distance that a "glorified" appendectomy removed all the diseased tissue. The second case is even more interesting. After a laparotomy performed on account of chronic appendicitis, Prof. Archibald, whose experience with such

TABLE XL.—ROENTGEN-RAY DIAGNOSIS FOR ULCERATIVE COLITIS.*

No. of cases.	Cases subjected to operation or autopsy.	Tuberculous colitis.
Negative		0 — 44
1726	46	+ — 2
Positive		0 — 0
869	62	+ — 62
Total 2595		

conditions is wide, found an extensive tuberculous colitis. The intestine was not opened, and the only explanation, that we have to offer, is that the disease had not at that time advanced to ulceration.

A more detailed study of the findings in the remaining 44 cases is of considerable interest. Many of them had moderately and far-advanced pulmonary tuberculosis and 2, who died from their pulmonary tuberculosis, were negative for tuberculous colitis at autopsy. In 1 patient, the colon was normal and 2 microscopic tubercles were

* See Tables 41 to 42 for operative and autopsy findings.

TABLE XLI.—CASES IN WHICH ULCERATIVE COLITIS WAS DIAGNOSED BY ROENTGEN-RAY

Case.	Pulmonary condition.	Roentgen classification.	Bacilli.	Tuberculous complication.	Location of barium shadows at the 6th-hour observation.						Spasm or filling-defect.	Location of barium shadows at the 24th-hour observation.					Enema spasm or filling-defect.	Site of ulceration from roentgen finding.	Site of tuberculous ulceration from operative or autopsy finding.
					Il.	Ce.	H. F.	T. C.	D. C.	R.		Ce.	H. F.	T. C.	D. C.	R.			
100	Min.	3	+	○	+	Sl. ○	+	+	..	..	Ce.	○	○	○	○	+	.....	Cecum	Ascending colon.
101	F. A.	3	+	○	+	Sl.	..	..	..	..	Ce.	○	○	○	○	○	Ce.–H. F.	Cecum—hepatic flexure	* Cecum.
102	F. A.	3	+	○	○	○	○	+	+		Ce.+	○	○	○	○	○	Ce.	Cecum +	Cecum—descending colon.
103	M. A.	3	+	○	+	+	+	+	..		Ce.–H. F.	○	○	○	○	○	.....	Cecum—transverse colon	Cecum.
104	M. A.	3	+	○	+	+	+	..	..		Ce.	○	+	+	+	+	Ce.	Cecum	Ascending colon.
105	M. A.	3	+	○	+	+	+	+	+		Ce.+	○	○	○	○	+	.....	Cecum—hepatic flexure	* Small bowel—colon. Autopsy.
106	F. A.	3	+	○	+	Sl.	..	..	..	..	Ce.	○	○	○	+	+	.....	Cecum—hepatic flexure	Ileum—cecum.
107	M. A.	..	+	○	+	Sl.	..	..	..	..	Ce.	○	○	○	+	+	Ce.+	Ileum—descending colon	Ileum—descending colon.
108	F. A.	3	+	○	+	..	..	..	..	..	Ce.+	○	○	○	Sl.	Sl.	.....	Cecum +	Cecum—transverse colon.
109	F. A.	3	+	○	+	+	+	+	+	+	Ce.–T. C.	○	○	○	○	○	Ce.–T. C.	Cecum—transverse colon	Cecum—transverse colon.
110	F. A.	..	+	○	+	+	○	+	+	..	H. F.	○	○	○	○	Sl.	.....	Hepatic flexure	Hepatic flexure.
111	F. A.	..	+	Nephritis	+	Sl.	..	..	..	..	Ce.	No	exa	min	atio	n	.....	Cecum +	Ileum—cecum.
112	F. A.	3	+	Larynx	12	hr. o	bser	vati	on		.....	○	○	○	○	+	Ce. ?	Cecum +	Ileum—cecum.
113	Min.	2	+	○	○	Sl.	+	+	+	+	Ce.	Sl.	Sl.	Sl.	Sl.	Sl.	.....	Cecum	Cecum.
114	Min.	3	?	○	+	+	+	+	+	+	Il.–T. C.	○	○	○	○	○	Ce.–T. C.	Ileum—transverse colon	Cecum +.
115	M. A.	3	+	○	+	Sl.	+	+	+	..	Ce.	○	○	○	○	○	Ce. ?	Cecum	Cecum.
116	M. A.	3	+	○	+	?	..	..	..	..	Ce.	○	○	○	○	+	○	Cecum	Ascending colon.
117	F. A.	1	–	Larynx	+	+	+	+	+	..	Ce.	○	○	○	○	○	.....	Cecum	Ascending colon.
118	F. A.	..	+	Larynx	+	+	+	+	..	..	Il.–Ce.	○	○	○	+	+	.....	Ileum—cecum	Cecum. Autopsy.
119	F. A.	3	+	○	+	+	+	+	+	+	Ce.	○	○	○	○	○	.....	Cecum—hepatic flexure	Cecum.
120	F. A.	3	+	○	+	+	+	+	+	+	Ce.	○	○	○	○	+	Ce.	Cecum.	Cecum. Autopsy.
121	F. A.	3	+	○	+	+	+	+	+	+	Ce.	○	○	○	○	○	.....	Cecum—hepatic flexure	* See table. Autopsy.
122	F. A.	3	+	○	+	+	+	+	+	+	Ce.	○	○	○	○	○	.....	Cecum	Cecum. Autopsy.
123	F. A.	3	+	Larynx	+	+	+	+	+	..	Ce.	No	exa	min	atio	n	.....	Cecum—hepatic flexure	Ileum—descending colon.
124	..	3	○	○	+	○	○	○	○	○	Ce.+	○	○	○	○	○	Ce.–Sig.	Non-tube. colitis	Non-tube. ulcerative colitis.
125	F. A.	3	+	Empyema	+	○	+	..	..	..	Ce.–H. F.	○	○	○	○	○	.....	Cecum—transverse colon	* Cecum — hepatic flexure. Autopsy.
126	F. A.	..	+	○	+	+	+	+	+	–	Ce.	No	exa	min	atio	n	.....	Cecum—hepatic flexure	Cecum—hepatic flexure.
127	F. A.	3	+	Ischio-rectal abscess	+	Sl.	Sl.	○	+	+	Ce.–H. F.	○	○	○	○	+	.....	Cecum—transverse colon	Cecum—transverse colon.
128	F. A.	..	+	.....	+	+	+	+	+	..	Ce.	○	○	○	○	○	.....	Cecum—transverse colon	Cecum—transverse colon.
129	F. A.	3	+	○	+	+	+	..	..	..	Ce.	○	○	○	○	○	Ce.	Cecum—hepatic flexure	* Mammillations in proximal colon (see table). Autopsy.

130	..	3	+		○		+	+	+	+	+	..	Ce.	○	○	○	○	○	.....	Cecum	Cecum.
131	..	3	+		○		+	+	+	+	+	..	Ce.	○	○	○	○	○	.....	Cecum	Cecum—hepatic flexure.
132	M. A.	..	+		○		+	..	..	..	..	..	Ce.	Sl.	+	..	..	..	.....	Cecum	Cecum.
133	F. A.	..	+		○		○	Sl.	+	+	+	..	Ce.	No	exa	min	atio	n	○	Cecum	Ileum—cecum.
134	F. A.	..	+	.....			○	+	+	+	○	+	Ce.	○	○	○	○	+	Ce.	Cecum	Ileum—cecum.
135	F. A.	..	+	.....			+	+	+	+	..	..	Ce.	○	○	○	○	+	Ce.	Cecum	* Ileum—colon.
136	F. A.	3	+	.....			+	Sl.	Sl.	Sl.	+	..	Ce.–T. C.	○	○	○	○	○	.....	Cecum—transverse colon	Cecum—transverse colon.
137	M. A.	..	+		○		+	+	+	+	..	..	Ce.	○	○	○	○	○	.....	Cecum—hepatic flexure	Cecum—transverse colon.
138	F. A.	3	+	.....			+	+	+	..	..	..	Ce.	○	○	○	+	+	Ce.	Cecum	Cecum.
139	..	3	−	.....			+	..	..	..	..	..	.....	○	○	○	○	○	.....	Cecum +	Cecum +.
140	M. A.	..	+		○		+	..	..	..	..	..	Ce.	○	○	○	+	+	.....	Cecum—hepatic flexure	Ileum—cecum.
141	M. A.	2	+		○		+	+	+	+	+	..	Ce.	Sl.	Sl.	○	○	Sl.	Ce.	Cecum	Cecum.
142	F. A.	..	+	.....			+	+	+	+	+	..	Ce.	○	○	○	○	○	.....	Cecum—transverse colon	Cecum +.
143	..	3	−	.....			+	+	+	+	+	..	Ce.–H. F.	○	○	○	○	+	Ce.–H. F.	Cecum—transverse colon	Cecum—transverse colon.
144	F. A.	3	+		○		+	+	+	+	+	..	Ce.	No	exa	min	atio	n	.....	Ileum—transverse colon	* Jejunum—rectum. Autopsy.
145	..	3	−	.....			+	+	+	+	..	..	Ce.–H. F.	○	○	○	+	+	○	Cecum +	Cecum—transverse colon.
146	Min.	3	○		○		+	+	+	+	+	..	Ce.–H. F.	○	○	○	○	+	Ce.	Ascending colon	Ascending colon.
147	F. A.	3	+		○		+	+	+	..	..	..	.....	+	+	+	+	+	.....	Ileum	* Ileum—ulcer in cecum. Autopsy.
148	M. A.	..	+		○		+	○	+	+	+	+	Ce.	○	○	○	+	+	.....	Cecum—hepatic flexure	Cecum.
149	M. A.	3	+		○		+	+	+	+	+	+	Ce. ?	○	+	+	+	+	.....	Cecum	* Ileum — transverse colon. Autopsy.
150	M. A.	3	+		○		+	+	+	+	+	+	Ce.	○	○	○	+	+	.....	Cecum—hepatic flexure	* Small bowel — colon. Autopsy.
151	F. A.	3	+		○		+	+	+	+	+	..	Ce.	○	○	+	+	+	.....	Cecum—hepatic flexure	* Duodenum—descending colon. Autopsy.
152	..	3	..	.....			+	+	+	+	..	..	H. F.	○	+	+	+	+	H. F.	Hepatic flexure	Ascending colon.
153	F. A.	3	+	Larynx			+	+	+	+	+	+	Ce.	○	○	○	○	○	.....	Cecum	Cecum.
154	N-T.	..	○		○		+	+	+	+	..	..	T. C. +	○	○	○	○	+	.....	Distal colon	Non-tubc. colitis.
155	N.-T.	..	○		○		+	+	+	+	+	..	H. F. +	○	○	○	○	○	.....	Colon	Non-tubc. colitis.
156	N.-T.	..	○		○		+	+	+	+	+	..	T. C. +	○	○	○	○	○	.....	Colon	Non-tubc. colitis.
157	F. A.	3	+	Larynx			+	+	+	+	?	..	Ce.–H. F.	○	○	Sl.	○	+	.....	Ascending colon	Cecum-ascending colon. Autopsy.
158	F. A.	3	○		○		+	+	+	+	+	+	Ce.–H. F.	○	○	+	+	+	.....	Cecum—hepatic flexure	* Ascending colon—sigmoid. Autopsy.
159	F. A.	3	+		○		+	+	+	+	..	..	Ce.–H. F.	○	○	○	Sl.	Sl.	.....	Ascending colon	* Ascending colon—sigmoid. Autopsy.
160	F. A.	3	+	Larynx			+	+	+	+	..	..	Ce.	○	+	+	+	+	.....	Cecum	Cecum. Autopsy.
161	F. A.	3	+	Larynx			+	+	+	+	..	..	Asc.	○	○	+	+	○	.....	Ascending colon	Cecum—splenic flexure. Autopsy.

* See Table I for a more detailed pathological finding.

TABLE XLII.—CASES IN WHICH INTESTINAL TUBERCULOSIS WAS EXCLUDED BY ROENTGEN-RAY

Case.	Pulmonary condition.	Roentgen classification.	Bacilli.	Tuberculous complication.	Location of barium shadows at the 6th-hour observation.						Spasm or filling-defect.	Location of barium shadows at the 24th-hour observation.					Enema. Spasm or filling-defect.	Operation or autopsy findings.
					Il.	Ce.	H. F.	T. C.	D. C.	R.		Ce.	H. F.	T. C.	D. C.	R.		
1	F. A.	3	+	○	(18th hour examination only)						○	+	+	..	..	..	○	Chronic tubc. appendicitis.
2	M. A.	1	○	○	+	+	+	..	..	..	○	+	+	+	+	+	.....	Ovarian cyst.
3	N.-T.	○	○	○	+	+	+	+	..	..	○	+	+	+	+	+	.....	Chronic appendicitis.
4	?	○	○	○	+	+	+	..	..	..	○	+	+	+	+	+	.....	Non-tubc. colitis.
5	M. A.	3	+	○	+	+	+	..	..	..	○	+	+	+	+	+	○	Chronic tubc. appendicitis.
6	M. A.	2	+	○	+	+	+	+	..	..	○	+	+	+	+	+	○	Chronic appendicitis.
7	M. A.	3	+	○	+	+	+	+	..	..	○	Sl.	○	○	+	+	○	Tubc. salpingitis and peritonitis. No tubc. colitis.
8	M. A.	3	+	○	+	+	..	..	..	..	○	+	+	○	○	+	.....	Non-tubc. appendicitis.
9	N.-T.	1	○	.....	+	+	+	+	..	..	○	+	+	+	+	+	.....	Gastric varicose veins.
10	M. A.	1	+	○	+	+	+	..	..	..	○	+	+	○	○	+	?	Non-tubc. appendicitis.
11	F. A.	3	+	○	+	+	+	..	..	..	○	+	+	+	+	+	.....	No tubc. colitis.
12	F. A.	3	+	○	+	+	+	+	+	..	○	○	+	○	○	○	.....	Chronic appendicitis.
13	F. A.	3	+	○	+	+	+	..	..	..	○	+	+	+	+	+	○	Extensive tubc. enteritis and colitis (see text).
14	Min.	1	○	○	+	+	+	Sl.	..	..	○	+	+	+	+	+	○	Chronic appendicitis.
15	M. A.	2	+	○	+	+	+	..	..	..	○	+	+	+	+	+	○	Chronic appendicitis.
16	N.-T.	–	–	○	+	+	+	..	..	..	○	+	+	+	..	..	.....	Gastric ulcer.
17	..	–	–	○	+	+	+	..	..	..	○	+	+	+	+	..	○	Gastric ulcer.
18	N.-T.	–	○	.....	+	+	+	+	..	..	○	+	+	+	+	+	.....	No tubc. colitis.

| | | |

present in the small bowel; in another, the colon was normal with 4 small, fresh ulcers in the terminal ileum; and in 1 case, an acute appendicitis. In 20 cases, a simple chronic appendicitis was found, in 3 a tuberculous appendicitis, in 2 gastric ulcer, in 1 each, ovarian cyst, tuberculous salpingitis and peritonitis, "gastric varicose veins," and Jackson's membrane. Two cases were described as normal, and 1 had completely healed ulcers in the ileum, and 7 were stated to have no tuberculous colitis.

One negative case (No. 42) is of special interest as at autopsy the larynx, the sternum, both lungs, the sacro-iliac joint, the right knee, the kidneys, the epididymis, prostate, seminal vesicles, and the meninges, were all tuberculous. That the cecum and colon should be spared, while the small bowel presented completely healed ulcers, is striking. The patient had had ultraviolet-ray treatment for some months.

TABLE XLIII.—PATHOLOGICAL DIAGNOSIS OF CASES IN WHICH TUBERCULOUS COLITIS HAD BEEN EXCLUDED BY ROENTGEN-RAYS.

		Cases.
Acute appendicitis		1
Chronic appendicitis		23
Simple	20	
Tuberculous	3	
Ovarian cyst		1
Tuberculous salpingitis and peritonitis		1
"Gastric varicose veins"		1
Gastric ulcer		2
Jackson's membrane		1
Normal		4
No tuberculous colitis		7
2 microscopic tubercles in small bowel		1
Completely healed ulcers in ileum		1
Extensive tuberculous enteritis and colitis		1
Small ulcer in cecum		1
4 small fresh ulcers in ileum		1
		46

It is of interest to note that, in the 62 cases diagnosed as positive by the roentgen-ray method, which went to operation or autopsy, all had tuberculous colitis. It is of further interest that of 5 cases, diagnosed as tuberculous enteritis (tuberculosis of the small bowel), 4 were found positive at operation and 1 at autopsy.

In the list of positive cases (see Table XLI) attention should be drawn again to the fact that, while only the findings at the sixth or seventh and the twenty-fourth or twenty-fifth hours have been noted, it is highly important to study them at the eighth and ninth hours as well. As a matter of fact, the sixth-hour observation may

be omitted. Under the head of "Site of Ulceration," we would not wish it understood that the intestine is affected only at the location mentioned, but rather that in our opinion a lesion existed at this location and possibly elsewhere. Furthermore, the time that elapsed between the roentgen-ray observations and autopsy, was usually many months and, in some cases, several years. Many such cases would, of course, have changed markedly during this lapse of time.

The two tables (Nos. XLI and XLII) demand very careful study, for upon the evidence, which they present, the diagnosis of intestinal ulceration is based. We have omitted purposely the observations at the eighth and ninth hours, but we would like again to call attention to the fact that a positive finding at the sixth or seventh hour must be confirmed at one or more subsequent observations.

Under the column, which indicates the position of the barium shadows at the sixth or seventh hours, the plus sign means that the head of the column has reached this location. When the space is left blank it implies that the head of the column has not reached this position. The sign, "o," indicates that the barium, which has reached this site, has passed on, and this portion of the colon is empty at this observation. The abbreviation, "Sl," means that this portion of the tract is not completely filled.

We would suggest that a careful comparison of the findings at the sixth hour in the table of the positive cases be made with those in the table of negative cases. The most striking thing in the table of the negative cases is the presence of the barium in the ileum, cecum, and hepatic flexure at six hours. Occasionally it is seen beyond this point, even as far as the splenic flexure, and, in 1 case in the rectum.

A study of the table of positive cases will show either a retardation or a hurrying of the passage of the barium through the large bowel, as indicated by the retarded or advanced position of the head of the column. This indicates, first, that the barium is retarded at the ileo-cecal sphincter, or, second, that it is hurried in many instances so far along the colon, that it is, at the sixth hour, in the transverse, or descending colon or even in the sigmoid and rectum.

As a result of the absence of barium in, or of a slight, partial filling of the cecum or ascending colon there is produced a "filling-defect," which is noted in the column under that heading. In a number of cases, for instance, Case 105, the observation at the sixth or seventh hour indicates that there was more than a slight amount

of barium contained in these segments. However, the spasm or filling-defect was definite, so that the presence of considerable barium does not necessarily signify absence of the characteristic finding of ulceration. A marked difference is seen in the positive and negative cases as a group at the twenty-fourth or twenty-fifth hour. The "o," as stated above, indicates that the barium has passed beyond this point. It will be seen that there is a decided hypermotility in the positive cases, whereas the observations in the negative group indicates an average or normal rate of flow.

Barium enemas administered in cases where it was indicated, are tabulated in the next column, where persistent filling-defect or repeated spasm, if it occurred, is noted. A comparison of the observations of the enema in the two groups is noteworthy. In the positive group note Cases Nos. 112, 116, 133, 145, where the enema failed to reveal definite filling-defect or spasm, and in the negative group Cases Nos. 19 and 24, which showed a filling-defect or spasm. Cases Nos. 24, 112 and 133 are illustrated on pages 202, 201, 200 respectively.

DIFFERENTIAL DIAGNOSIS OF INTESTINAL TUBERCULOSIS.

Chronic Appendicitis.—In the experience of the authors, a diseased appendix, even though it be tuberculous, has not produced the localized or generalized hypermotility with filling-defects, similar to those seen in tuberculous ulceration. However, adhesions, that may develop in consequence of an inflamed appendix, can very easily deform the outline of the cecum and should be borne in mind.

Malignant Tumors.—Carcinoma or, in fact, any malignant process may produce a filling-defect very similar to that obtained in tuberculous colitis, and, in its earlier phases, after ulceration, may produce also the hypermotility. But, as the wall of the gut becomes more involved, a stenosis is usually produced, which would tend to slow the flow. In many cases, an outstanding manifestation is the obstructive symptoms that are present, as well as a definite proximal stasis, often with dilatation. This latter is exactly the opposite to that observed in tuberculous ulceration, where there is rarely any proximal delay or stasis. With the use of the barium enema, there is usually noted a permanent defect.

Amœbic Dysentery, Sprue and Non-tuberculous Ulcerative Colitis apparently produce the same shadow complex as that seen in tuberculosis, and it may be impossible to differentiate them. Fortunately many of the forms of non-tuberculous colitis more commonly affect

FIG. 61 (Case 5).—Roentgen finding at eight hours: Slight gastric retention; irregular and fixed appendix (*APP.*); no filling-defect of cecum. Confirmed at six and seven hours.

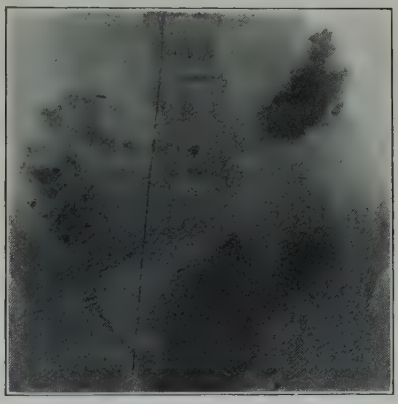

FIG. 62 (Case 5).—Roentgen finding at twenty-four hours: nothing definite.

FIG. 63 (Case 5).—Roentgen finding after enema: slight irregularity of the cecum (*I*): no filling-defects; incompetent ileo-cecal valve.

Operative Finding.—See Case 5. Table XXXVII.

Clinical Finding.—Male, single, aged twenty-nine years. Pulmonary tuberculosis began December, 1915. In April, 1917, infiltration right upper and left lung. No intestinal symptoms at this time. In 1920, had symptoms referable to chronic appendicitis. Roentgen-ray confirmatory. In May, 1920, operation and chronic tuberculous appendix removed. Later, gastric ulcer diagnosed. Well in 1924.

FIG. 64 (Case 5089).—Spastic colon. Roentgen finding at six hours: head of the column in the sigmoid; the haustra are irregular and deep (pseudo-filling-defect) (*P.F.D.*); considerable gas bubbles, but no localized spasm as seen in ulceration.

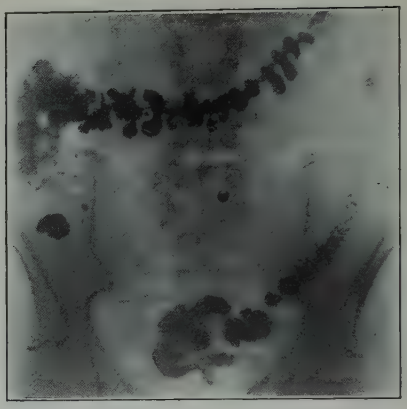

FIG. 65 (Case 5089).—Roentgen finding at twenty-four hours: note the irregular deep haustra ("spastic colon") but with the average residual that occurs in normal cases.

Clinical Finding.—Male, single, aged forty years. Fibroid tuberculosis. Right and left upper one-third. Not very acutely ill. Condition favorable on admission and discharge. Later, operation for hyperthyroidism. No abdominal symptoms. Has remained well for four years.

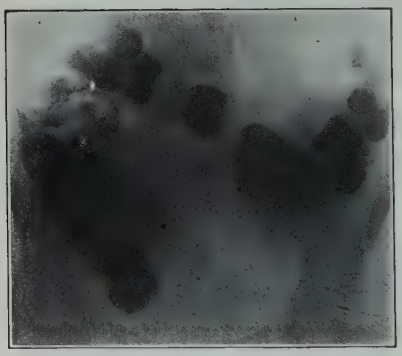

FIG. 66 (Case 5183).—Roentgen finding at six hours: somewhat advanced position of the head of the column (hypermotility); no filling defects.

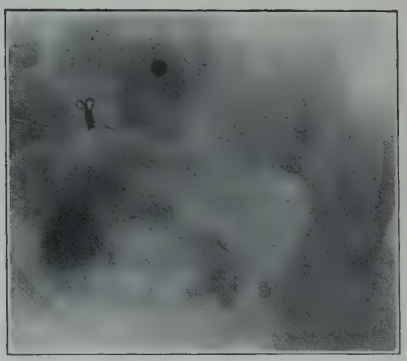

FIG. 67 (Case 5183).—Roentgen finding at twenty-four hours: general hypermotility, note most of the barium remaining is in the ascending colon; no filling-defects, just the opposite to that which is found in tuberculous ulceration.

Clinical Finding.—Female, married, aged thirty years. Pulmonary tuberculosis doubtful. Thyroid enlarged. Had severe diarrhea, eight stools a day. Marked abdominal symptoms. Has remained well for four years.

FIG. 68 (Case 9227).—Effect of castor oil. Roentgen finding at two hours: stomach well filled; the head of the column probably near the ileo-cecal sphincter.

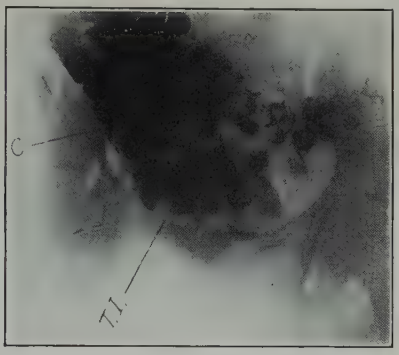

FIG. 69 (Case 9227).—Roentgen finding at six hours: stomach completely empty; terminal ileum fairly well filled (*T.I.*); the head of the column somewhat advanced; note the colon is well filled to this site, particularly the cecum (*C*).

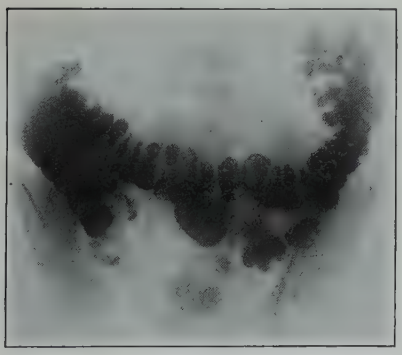

FIG. 70 (Case 9227).—Roentgen finding at nine hours: ileum practically empty; head of the column has moved forward considerably (now in the sigmoid); note the proximal colon is still well filled.

FIG. 71 (Case 9227).—Roentgen finding at twenty-four hours: hypermotility; note the residual in the cecum (normal) and compare with Fig. 36 (pathological cecum).

Clinical Finding.—Male, married, aged forty years. Slight pulmonary tuberculosis. At times marked nervous symptoms. No hyperthyroidism. Has remained well for eight years.

the distal portions of the colon. The lesions, however, may extend as far back as the cecum and often produce, when studied by enema, a picture, resembling a non-haustrated tube, very different from the repeated spasms or filling-defects of the cecal region, seen in ulcerative colitis. Amœbiasis may begin, of course, in the cecum. We have found no definite data on actinomycosis.

Diverticulitis.—Diverticulitis is characterized by a circular spotted effect, produced by the barium remaining in the diverticula, following the ingestion of a meal or after the administration of an enema. Hypermotility may be present.

Achylia Gastrica and Hyperthyroidism.—Achylia gastrica, hyperthyroidism and possibly other conditions may bring about a general hypermotility, but have failed, as far as we know, to reveal filling-defects and localized spasm.

Jordan describes in detail a number of conditions in which increased peristalsis may be observed, among them cases of diarrhea, whether accidental or habitual, colitis, and patients taking liquid paraffin. These are fully illustrated and it is interesting to note, none shows the typical picture that is seen in tuberculous ulcerative colitis. Another point of great importance is that, in a few of his cases of colitis, there is a cecal retention of twenty-four hours or more and, in 1 case of marked colitis, a retention in the proximal colon of thirty-six hours. This is diametrically opposite to the findings in tuberculous ulceration.

A series of cases were examined following the ingestion of a cathartic (castor oil) and, while there was a decided increase in the rapidity of the passage of barium through the large bowel, the colon did not present the same manifestations, localized filling-defects and spasm, which occur in those patients proved to have tuberculous colitis. The former cases showed smooth haustrations, and, while the contents moved along more rapidly than they did when not under the influence of the cathartic, a spastic or ragged contour was never visualized.

CHAPTER XXIV.

COMPLICATIONS OF INTESTINAL TUBERCULOSIS.

The frequency of complications in intestinal tuberculosis depends, in part, upon the time at which the diagnosis is made and treatment begun. It is striking how few complications are noted clinically, when proper treatment is instituted early and many may escape all complications. But, in a certain number of instances, in spite of any and all forms of treatment, the combined tuberculosis of the lungs and intestine steadily advances to death. During this period of slow progression, several complications and sequelae have been noted as less uncommon than others and among these are stenosis, perforation of the ulcer or ulcers, with generalized peritonitis or fecal abscess as an end-result, severe intestinal hemorrhages, involvement of the mesenteric glands leading at times to severe and persistent pain. The following tabulation of complications is based upon Maylard's.

COMPLICATIONS AND SEQUELÆ OF ULCERATIVE INTESTINAL TUBERCULOSIS.

1. Mixed infection.
2. Hemorrhage.
3. Stricture.
   - (*a*) From cicatrix of healed ulcer.
   - (*b*) From cicatrix of tuberculous granulation tissue (fibrous).
   - (*c*) From hyperplastic tissue formation of active ulcer.
4. Localized tuberculous peritonitis.
   - (*a*) Causation of adhesions.
   - (*b*) Formation of localized effusions.
   - (*c*) Formation of localized abscess.
   - (*d*) Formation of fistulæ, either
     1. Into a neighboring viscus.
     2. Onto the surface of the body.
5. Generalized tuberculous peritonitis.
6. Perforation into the general peritoneal cavity.
7. General miliary tuberculosis or advanced lesion in other organs.
8. Amyloid disease.
9. Intussusception.
10. Carcinoma.

SECONDARY INFECTION.

Maylard has laid considerable stress upon mixed infections in intestinal tuberculosis. Such conditions, he thinks, not uncommon, especially in ulcers of the lower ileum and cecum, where, from prolonged contact with the feces, such conditions would be more likely to occur. Infected ulcers are very irregular with ragged, gnawed edges. Perforation and abscess formation are more likely to occur. Such ulcers are accompanied more frequently than others by rise of temperature and marked tenderness. Ophüls, in a careful study of 22 cases of intestinal tuberculosis, attempted to verify Orth's statement that non-specific ulceration (*i. e.*, due to other organisms) as well as tuberculous ulceration could occur in the same patient. In only 3 cases could other bacteria be connected with the ulcerative process.

Gant states that intestinal tuberculosis may be complicated by amœbic, bacillary, balantidic, luetic, or helminthic, ulcerative colitis or colitis due to mixed infections. Godel reports 4 cases, coming to autopsy, of chronic amœbic dysentery and tuberculous enterocolitis. Amyloid disease of the walls of the intestinal arteries (in the bowel wall) may produce ulceration. Rolleston thinks pyococci play an important part both at onset and later.

INTESTINAL HEMORRHAGE.

Macroscopic blood in the stools of patients with advanced intestinal tuberculosis is by no means common, but occult blood occurs even in many early cases where macroscopic blood is wanting (see p. 170). Degail, in his excellent thesis, discusses this subject under the heads of black diarrhea, streaky hemorrhages, dysenteric form, and abundant hemorrhages, at times rapidly fatal. Black diarrhea, really due to blood pigmentation, occurs usually late in the disease and has the appearance of coffee grounds. If the stools are frequent, erythrocytes can usually be found. Girode found them in 13 of 22 cases. Guyénet divides the cases into 2 groups: first, mechanical due to rupture; second, diapedesis. The bleeding vessel is usually difficult to find at autopsy and, while Cruice failed to find it in any of his 4 cases, Hanot found a bleeding vessel the size of a goose feather in the cecum at autopsy on a man, aged about fifty-six years, who had passed unconsciously 2 liters of blood and feces. Atkin, in 1900, reported a case of fatal hemorrhage of the bowels in a woman, aged about thirty-seven years, with intestinal tubercu-

losis, stenosis, obstruction and vomiting of blood. At necropsy, 4 large tuberculous ulcers were found in the bowel, which was free of blood. The stomach was normal. Thus, severe and even fatal hemorrhages, first noted by Tonnelé in 1829, do occur but are rare. Only 1 fatal case has come under our observation and the hemorrhages extended over several days. Guyénet, in 1892, collected 15 cases and, in 1913, Cruice added 10 more. From a study of 1620 cases at the Phipps Institute, Cruice found 4 in which over 300 cc. of blood were lost and 20 others which lost up to 68 cc., including 2 with purpura hemorrhagica and 1 with simple purpura.

Chandèse has reported 4 fatal cases, Hanot, 3; Percy Kidd and Engelsmann 2 each; Austin Flint, Bounaix, Knoblauch, Cruchet and Magne (in a girl aged twelve years), Reimer, Vallin, Eakin, Maylard, J. Mueller 1 each.

Lemon, in a series of 50 cases, had none with intestinal hemorrhage.

Eakin reported the case of a male patient, aged fifty-four years, stableman, admitted to the hospital September 21, 1921. Six days after admission, he complained of pain in the rectum and passed a small amount of blood. Rectal examination was negative. Three days later, he lost 10 ounces of blood; the following day, one ounce; and, on the succeeding day, a large quantity. No abdominal tenderness or distention was present. Following this the patient grew rapidly worse and died October 14, one hundred and thirty-six days from onset. At autopsy, with the exception of the sigmoid and rectum, which contained blood-stained feces, the intestines were collapsed. There was a solitary ulcer in the mid-portion of the transverse colon, transversely 4 cm. in diameter, and situated opposite the mesenteric attachment. It contained neither blood nor blood clot. The serous surface opposite showed no gross evidence of tuberculosis. No other ulcers were found in the gastro-intestinal tract. Numerous tuberculous cavities were present in the right upper lobe.

Flint reports a case of a male, aged twenty-five years, an engineer, who had occasional attacks of rheumatism but was always well until six weeks before admission, when he began to cough. Diarrhea began ten days before admission and, five days before admission, he passed blood from the bowels in large quantity and fainted from the hemorrhage while at stool.

On the second day after admission, he had another large hemorrhage, fainted from the loss of blood, and died the next day. At autopsy, there was a cavity at the right apex and many ulcers in the colon and rectum, a few in the ileum, and a perforation.

Levinson reported a case in which three days before death, large quantities of clotted blood were found in the stools of a patient with slight pulmonary and extensive glandular and intestinal tuberculosis. At autopsy, an ulcer in the splenic flexure was found to have ruptured into the spleen, which was filled with blood clots, weighed 1420 gm., and filled the entire left side of the abdomen. Death was due to hemorrhage from the spleen into the colon.

In severe cases, the usual symptoms of intestinal hemorrhage present themselves, pallor, rigor, rapid short respiration, small pulse, subnormal or falling temperature, cold extremities and surfaces, cold sweats and syncope. Death more frequently occurs, as in our case, after several days of bleeding. The bleeding may come from ulcers in the small or large bowel. It is usually dark (tarry), intimately mixed with the feces, and malodorous, when from the small bowel; reddish, often clotted, or even on the surface of feces, when from the large.

It should not be overlooked that, in older patients, bleeding from the bowels is usually due to malignant disease of the colon, which can, though rarely does, coexist with tuberculosis.

The prognosis is usually very bad for patients with severe hemorrhage, but Peters and Bullock have reported 2 recoveries and Bonney 1. The best treatment is rest, morphine, ice to the abdomen, abstention from food at first and later liquid diet. Fibrogen (Mills) may be tried. In chronic hemorrhagic colitis lavage after appendicostomy has been effectual. (See castor oil, p. 262.)

PERFORATION.

Attention has been called to the fact, that the first effect of ulceration of the bowel on the peritoneum is to cause a localized peritonitis (serositis) which may produce adhesions with neighboring organs. Microörganisms may pass through such an injured peritoneum and abscesses arise or, indeed, if the adhesions yield, general peritonitis.

Complete or partial perforation of a tuberculous ulcer can result then in peritonitis or fecal abscess. The clinical picture of such a case of peritonitis in advanced pulmonary tuberculosis is noteworthy for its lack of symptoms. It can be entirely overlooked which occurred in one of our patients. Usually, however, a sudden and severe pain, following a feeling of a snap or break, is noted at the time of rupture, which may occur while the patient is at stool, while walking, or during retching. The patient may die rapidly from collapse, or may continue to complain of pain in the epigas-

trium or the right iliac fossa, which is increased by pressure or movement. The temperature may be subnormal and the abdomen rigid. Nausea, vomiting, increased rapidity of breathing, some elevation of temperature, and a quickened pulse may occur with, in a few cases, abdominal distention. The presence of tuberculous peritonitis may mask the symptoms. Some of the deaths from exhaustion and asthenia may be due to this, usually to the great surprise of the clinician (Austin Flint).

Adami and McCrae, in 85 autopsies on patients dying of pulmonary tuberculosis, found 2 with perforation. In his 100 cases, Engelsmann found the peritoneum involved in 22, 13 times with exudate and 9 times with caseous adhesions. In only 2 was the condition recognized clinically. In 475 autopsies on patients dead of pulmonary tuberculosis, Cruice found 13 cases of perforation, 10 of them complete, occurring 6 times in the small and 4 in the large bowel (appendix 3, cecum 1). Four gave the typical symptoms, already described, and 5 had indefinite symptoms (1 pain only, 1 a drop in temperature only, 2 no abdominal symptoms). One case had four perforations and 2 two each. Multiple perforation is by no means rare in these cases, where, in 1 instance eight, and in another eleven, were found.

Bérard and Patel reported 26 cases of general peritonitis, following perforation of tuberculous ulcers. O. Mueller found 13 cases of perforation in 57 cases of intestinal tuberculosis in children. Korbacher has reported a case with thirty perforations in 8 to 10 fist-sized sacs.

Kenéz has described a case of perforation, where twenty-four hours before death, gas was discovered by roentgen-ray examination under both sides of the diaphragm.

Albrecht (quoted by Pankow), in a study of 1348 male and 807 female patients dead of tuberculosis, found intestinal tuberculosis in 49 per cent of the male and 51 per cent of the female, peritonitis in 10.46 per cent of the male and in 10.89 per cent of the female. The peritonitis was combined with intestinal tuberculosis in 42.4 per cent of the male, in 39.5 per cent of the female. In Aschoff's laboratory tuberculous peritonitis was found combined with intestinal tuberculosis in 53.49 per cent of the males and in 65.78 per cent of the females. The origin of the tuberculous peritonitis was the intestinal tuberculosis in 12.6 per cent of the male and in 7.7 per cent of the female.

Maurange, in 107 cases of tuberculous peritonitis, noted intestinal tuberculosis in 80.

Three fatal cases of perforation have come under our notice, 2 of which were diagnosed clinically. Gardner, in 51 autopsies on patients with intestinal tuberculosis, had 3 fatal cases, due to perforation of the ileum in 1 case, of the appendix in 1, and of the cecum in 1. Letulle's experience agrees with Gardner's, for he has rarely observed peritonitis in intestinal tuberculosis but localized peritonitis is common. (See p. 86).

FECAL ABSCESS.

Fecal abscess may produce few symptoms. Nausea and vomiting may occur with some local pain and bloody stools. On abdominal examination a tumor is usually found, most often in the right iliac fossa, with irregular and ill-defined outlines, immovable on deep respiration or on pressure, tympanitic on light, dull on deep percussion, with gurgling and pain on pressure or resonance suddenly replacing previous dulness (said to be pathognomonic). A splash may be obtained.

The tuberculous ulcer may perforate into other organs or on to the body surface and form fistulæ. One coil of the small bowel may rupture into another, either the large or the small may communicate with the stomach, the large may open into the small, the small bowel into the bladder.

The passage of intestinal gas or coloring matter is the only sure evidence of fecal fistulæ. Many of these cases in Maylard's experience, had shown previous symptoms of tuberculous peritonitis.

The "pyo-pneumothorax subphrenicus" of Leyden pushes up the diaphragm, compresses the base of the lung, but never displaces the heart laterally. Succussion is heard only in the abdomen, not in the chest, as occurs when the pleura is involved.

STENOSIS.

Attempts at healing of the tuberculous ulcers may result in the formation of much connective tissue, which may contract, narrow the lumen of the gut and bring about stenosis. Hypertrophy of the muscularis above the constriction may for a time overcome it, but, eventually, in many such cases symptoms arise. These may be only temporary and due to occlusion of the lumen by some particles of food, which eventually undergo digestion.

The symptoms of stenosis may be so slight as to attract no attention or, indeed, can even be entirely lacking and the condition

discovered only at autopsy. E. Fraenkel had a case with eight stenoses and no symptoms. There are no characteristic symptoms. Often the symptoms, which may always be slight, are a little pain after food, which does not occur regularly and, only after one or two years, shows signs of increasing. As the stenosis increases, the pain occurs more regularly after each meal, becomes colicky, borborygmi are heard, and the patient complains of much gas and shortness of breath. In severe cases definite signs of obstruction supervene with nausea, fecal vomiting and, unless surgical interference is called upon, death. Such cases are rare. At times it is possible to palpate through the thin abdominal walls the intestinal coils which stiffen for several seconds during each spasm. Inanition, anemia, and cachexia, due to the stenosis, may lead to death.

Stenosis occurs most often between the twentieth and fortieth years. It is questionable whether females are affected more than males. It is much more common in primary intestinal tuberculosis.

The strictures occur most frequently in the ileo-cecum, ileum and jejunum, but can occur elsewhere. Wieting's collected figures concerning the site are: Duodenum, 1; jejunum, 1; ileum 74; ileo-cecum and ileum 6; ileo-cecum 79; cecum, 43. Caird found 14 in the ileum, 11 in the ileum and ileo-cecum, and 8 in the ileo-cecal valve. Nikoljski gives the following collected figures:

	Cases.
Small bowel	67
Cecum	30
Ascending colon	21
Transverse colon	4
Ileo-cecal region	36
Rectum	1

Felix collected 72 cases and found the site in the first third of the jejuno-ileum in 8.9 per cent, in the second in 17.8 per cent and in the last third in 73.2 per cent.

In 62 collected cases (Brunner), the stenosis was single in 24, multiple in 34. As many as seventeen (Jaboulay) have been described in 1 case and Strehl found thirteen in the small bowel, one at the ileo-cecal valve and the fifteenth in the ascending colon. Matas excised three strictures, found in 13 inches of the jejunum.

A stricture, following a girdle ulcer, may be annular and single or, as in Hofmeister's case, multiple, for he found ten ring strictures in 2.5 meters of bowel. Thirteen of his 83 cases were multiple. In other cases a bandlike bridge occurs. Maylard has described a kink-stenosis, where the peritoneal surfaces of two adjoining or of one large ulcer in the same loop became adherent. Between the

stenoses the small intestine may be so greatly dilated as to measure 9½ inches in circumference. In 1 case without symptoms of stenosis, the dilated sacs resembled stomachs and the fluid in them led to the diagnosis of peritoneal effusion (Litten). On several occasions the dilatations have been diagnosed "displaced stomach." Hypertrophy of the muscularis occurs above and atrophy of the wall below the stricture. Roentgen-ray study may fail to determine the site of the lesion or even indicate its presence elsewhere, as in a case of Schueppel's, where a spasm in the stomach was misleading. This observer called attention also to a spastic stenosis, due to irritation of the wall. In such cases, at autopsy or operation nothing can be found. He reported the eighth case on record of a stenotic tuberculosis of the jejunum.

While belladonna or atropin may give temporary relief, anastomosis, if the patient can stand such an operation, is the treatment to be preferred. The prognosis is far from good.

A fecal fistula or a small open sinus in a surgical scar, following an appendectomy, when actinomycosis is excluded, is very suggestive of intestinal tuberculosis (Lemon).

In Lemon's series of 50 cases, definite peritonitis was present in 10 and, among others complications were noted, appendicitis, gall-bladder disease, gastric ulcer, meningitis, pericarditis (tuberculous), Addison's disease, lupus, broncho-pneumonia, obstruction, multiple fistula and genito-urinary tuberculosis.

AMYLOIDOSIS.

The frequent association of amyloid or waxy degeneration with advanced pulmonary and intestinal tuberculosis demands some discussion. It has been stated that, in pulmonary tuberculosis, this condition is due always to a mixed infection in the lung or to intestinal tuberculosis (Largo). Engelsmann found it in the spleen, kidney and liver in 5 of his 100 autopsies and F. W. Zahn in 79 of 1528 cases of florid pulmonary tuberculosis. Holmgren has endeavored to connect two phenomena, watch-glass nails (as broad as they are long, curving equally lengthwise and sidewise) and thickening of the subcutaneous veins together with a narrowing of their lumen, on the forearms and legs, with the waxy degeneration. These conditions can occur in other diseases, but, when they occur in pulmonary tuberculosis, amyloid degeneration is found in 75 per cent of the cases, when they are absent, amyloid disease is

absent in 75 per cent of the cases. Albuminous urine with amyloid casts and a large hardened spleen are also occasionally found.

Herzog and Crowder have had cases of carcinomatous degeneration of a tuberculous ulcer and Caird has had 2 cases of the combined diseases. Stettin reports such a case. Naegeli reports a similar case with the carcinoma in a tuberculous ulcer of the ileum.

The involvement of the mesenteric glands rarely gives any clinical symptoms in secondary intestinal tuberculosis and is often only a "pathological sequence." In rare cases, they may cause severe abdominal pain. They are involved more frequently in children, where, in 27 cases with intestinal tuberculosis, Guthrie found them always diseased. Spontaneous healing can occur.

Invagination (intussusception) has been reported as occurring in 2 cases of intestinal tuberculosis and was attributed to the disease. Thorner has also reported a case of ileo-cecal intussusception complicated by intestinal tuberculosis.

CHAPTER XXV.

PROGNOSIS OF INTESTINAL TUBERCULOSIS.

The occurrence of persistent diarrhea in a patient with pulmonary tuberculosis has from time immemorial been considered of grave prognostic significance. "Diarrhea," wrote Hippocrates, "attacking a person with phthisis is a mortal symptom;" and again, "phthisical persons, the hair of whose heads falls off, die if diarrhea sets in." Such views have been held until very recently. From time to time, various pathologists have described attempts at healing of tuberculous intestinal ulcers, but they have usually stated that, while some ulcers tend to or do heal, others, in the same patient, show signs of advancement or, indeed, one and the same ulcer may show tendencies to heal and advance in different parts simultaneously. Until recently, it has been impossible to say when intestinal tuberculosis began in patients with pulmonary tuberculosis. Now, we are aware that the diagnosis, when made clinically, is usually made only when the disease in the bowels has reached an advanced stage, when, possibly, most of the chances of recovery are gone. In fact, prognosis of intestinal tuberculosis, until the third decade of this century, was in the same state that prognosis of pulmonary tuberculosis was in about 1870; it could be cured but a cure was a medical curiosity. As late as 1909, Walsh strongly urged against making a diagnosis, as such a diagnosis banished all hope of recovery from patient, nurse and physician. Such a view was logical at the time, for, when a diagnosis of intestinal tuberculosis could be made, the lungs were usually so riddled with disease that all hope was gone. In fact, if a suspicion of intestinal tuberculosis occurred to the physician on account of a stubborn diarrhea, abdominal pain and tenderness and, later, the patient recovered from such symptoms and arrested his pulmonary tuberculosis, it was considered proof sufficient that no intestinal tuberculosis had ever been present.

Recently a few have stated that certain cases of ileo-cecal tuberculosis may run a "less unfavorable" course, if the lungs or other affected organs do well, and a number have reported cures. However, Brauer and L. Spengler consider, if it is severe, that it is a contra-indication to artificial pneumothorax with which Forlanini

agrees, and an absolute contra-indication to thoracoplasty. Brunner says diarrhea is an incurable sign. We refer, of course, only to secondary intestinal tuberculosis in patients with pulmonary tuberculosis, for, in the primary form, in young growing persons and in the localized form in adults with favorable lung conditions, some have bespoken a more favorable prognosis. Kieffer thinks the higher in the bowel ulceration occurs, the worse the prognosis. Gant thinks that it is almost impossible to arrest patients with a deep ulcerative type. If staphylococci occur in the blood, J. Mueller thinks, the prognosis is especially bad.

The prognosis of tuberculous ulceration of the intestine in advanced phthisis is invariably bad, Schwatt thinks, and the onset of this complication ushers in the termination of the case within from three to six months.

Our experience leads us to hold in regard to prognosis in intestinal tuberculosis the same attitude that we maintain toward prognosis in pulmonary tuberculosis. If the general condition is good, if the pulmonary condition is in only a moderately advanced stage, if the intestinal lesion is apparently not extensive and if, finally, the patient can be placed under proper heliotherapeutic and general treatment, the prognosis for the intestinal tuberculosis is decidedly favorable. Many patients, under such conditions, lose permanently all intestinal symptoms and on roentgen-ray examination regain apparently a normal intestinal condition. Even when laryngeal tuberculosis accompanies such a condition, recovery may ensue. When, however, the pulmonary condition is far advanced, with cavities in the lung, and the general condition is poor, little can be hoped for, except amelioration of symptoms, and the alleviation, except by small doses of opiates, may be slight.

In such cases, the intestinal tuberculosis usually hastens the inevitable end. Prognosis, then, depends, as in pulmonary tuberculosis, upon early diagnosis and proper treatment, as intestinal tuberculosis usually only hastens the death from pulmonary tuberculosis. The statements of William Stokes in 1844 that "I never heard of a recovery after diarrhea had occurred," of Penzoldt that "intestinal tuberculosis makes pulmonary tuberculosis unhealable," of Behrend that "intestinal tuberculosis plays the final drama," are far from true in many cases today, for Gardner has shown that at autopsy on patients dying from pulmonary tuberculosis, many present evidence of healing, almost healed or indeed completely healed intestinal tuberculosis.

CHAPTER XXVI.

PROPHYLAXIS OF INTESTINAL TUBERCULOSIS.

Little can be said in regard to prophylaxis of intestinal tuberculosis. Warning about swallowing the sputum should always be given, but, in patients who affirm that they *never* swallow any sputum consciously, tubercle bacilli are still found in the stools. The saliva of some patients contains tubercle bacilli and, as every one swallows his saliva, it is impossible to prevent some tubercle bacilli from entering the intestines. Again some hold that tubercle bacilli gain entrance through the bile, over which, of course, we have no control. The danger of constipation, of allowing the feces to remain for long periods in contact with the walls of the cecum and large intestine has been emphasized. For this, as well as for other reasons, constipation should always, in patients with pulmonary tuberculosis, receive more careful treatment than has hitherto been accorded it. Irritative intestinal conditions should be remedied as quickly as possible. In all anacid cases, Kantor advises giving hydrochloric acid in full doses (♏ 20 to 30, in one-half to a glass of water, to be sipped with meals), basing his advice upon the observation, that tuberculous ulcers occur higher in the ileum in this condition (Rolleston). Inasmuch as intestinal tuberculosis, after all is said and done, depends directly upon the resistance of the patient, the prophylaxis of intestinal tuberculosis goes back again to the early diagnosis and arrest of the pulmonary tuberculosis before such a stage of lessened resistance is reached.

CHAPTER XXVII.

TREATMENT OF INTESTINAL TUBERCULOSIS.

The severe or even fatal prognosis given to patients with pulmonary, complicated with intestinal, tuberculosis since the time of Hippocrates, cannot be based upon the fact that tuberculous ulcers in the intestine never heal, for there is increasing proof at present to show that the contrary is the fact (see p. 67). But many, who have made a study of pulmonary tuberculosis, have long been dissatisfied and even impatient with the modern status of the treatment of secondary intestinal tuberculosis. The striking results, obtained in the treatment of laryngeal tuberculosis by vocal rest and in pulmonary tuberculosis by collapse therapy, suggested, of course, that resort to surgery might give the intestines rest and so bring about recovery. The diagnosis, when we started sending our patients to the surgeon, was made on clinical symptoms and abdominal examination. The outcome, of course, was, that the cases we referred for operation were usually far advanced and the results in many instances unsatisfactory, for patients with advanced pulmonary tuberculosis and advanced intestinal tuberculosis are necessarily poor surgical risks. This led us, however, to search for a means of earlier diagnosis, which has already been described in detail. Even with this method of detecting earlier cases, the results of surgical treatment were not satisfactory, and we turned to heliotherapy, which we had used in a few cases some ten or fifteen years previously. The general and medicinal treatment, which we had employed throughout this whole period, was constantly called upon to aid the other methods we used from time to time but it was, of course, only palliative.

GENERAL MEASURES.

In any patient with constant diarrhea and pain, steps must be taken at once to relieve such symptoms until the other measures employed become effective.

Protection of Abdomen.—In the first place, the patient should be made to wear an abdominal band of wool next the skin, which should be changed night and morning. These can be obtained in many large department stores and from any dealer who handles Jaeger

products. A piece of thick flannel, cut on the bias, and held in place with safety pins, is perfectly satisfactory and gives some patients more comfort than the bands. The flannel or band should cover the whole abdomen from the ensiform cartilage to the pubes. A more elaborately padded or quilted abdominal covering is preferred by some patients. Whatever the covering may be, it should be worn day and night and left off only while changing clothes or bathing. At night, a small pillow, shawl, or sweater may be placed over the abdomen on top of the night clothes, for the drop in temperature at night may upset or keep upset the irritable bowels.

Exercise.—As exercise may increase the peristalsis, it is a wise procedure to keep patients with pronounced diarrhea in bed until it ceases or lessens markedly.

The Diet.—The diet should be regulated until the diarrhea ceases, or until it is made certain that none of the food taken acts disadvantageously. Increased or active peristalsis may be caused by cold drinks (even cold water), milk, strong coffee, concentrated beef juice, fruit, fruit juices, salads and many vegetables. Tropon, somatose and raw eggs may act similarly in some patients and in these cases the eggs should be boiled for one minute and then taken like a raw egg. Boiling or warming the milk may prevent such unpleasant action. Fluids may have to be restricted. It should always be borne in mind, that the act of eating may produce reflex peristalsis in the terminal ileum and colon, independent of course of what is taken. Distention of the bowel is the natural stimulus of peristalsis and, when this is overactive, foods that cause fermentation, may produce or increase the diarrhea. They include among others, peas, beans, cabbage, turnips, potatoes, pastries prepared with yeast, rye bread, and at times milk and other foods.

Coarse food, which leaves much detritus, and especially raw fruit or cooked fruit, containing fine seeds, may cause slight but sufficient mechanical injury to increase the peristalsis. Raw salads may act similarly and, if very acid, may cause some chemical irritation of the ulcers.

A diet, exclusively of condensed milk (1 to 2 quarts a day), diluted with rice water, has been advocated by Mazen in all cases where the patient has not an absolute distaste for milk.

Certain drugs such as thiocol, which increase peristalsis should be avoided. Chilling of the feet is said to act disadvantageously in some patients.

It is striking that in patients, who respond favorably to treatment, how quickly almost every food can be eaten without any bad results.

HELIOTHERAPY.

Anyone, at all familiar with the treatment of tuberculosis, must have noticed for years in the literature references to the use of heliotherapy in intestinal tuberculosis. As the results that we and others had obtained from the surgical treatment of this complication had been far from encouraging, even in early cases, attention was turned to heliotherapy, in the hope that many early cases would respond with at least some alleviation of symptoms. Up to the time this method of diagnosis was described, it has been impossible, as we have pointed out elsewhere, to say when intestinal tuberculosis began or, indeed, even in many advanced cases, to decide whether or not it was present. This uncertainty as to diagnosis has without question been the reason why so little interest has been manifested in the subject, as it was just as impossible hitherto to determine the effect of any form of treatment as it was to decide definitely upon the presence or absence of the complication.

Some ten or fifteen years ago we treated several cases of what appeared to us at that time to be secondary intestinal tuberculosis, with natural heliotherapy. The patients showed some improvement but the uncertainty of diagnosis led us to discontinue the method. A visit to Leysin and to St. Moritz, where we saw the work of Bernhard and Rollier, and, later, the introduction of the quartz mercury-vapor lamp, made it seem advisable to push heliotherapy in the treatment of early intestinal tuberculosis. Living in a climate where it is impossible to use natural heliotherapy systematically, we were naturally forced to use the quartz mercury-vapor or the carbon-arc lamp. In our experience the former is much simpler and can be controlled more easily by patients, who administer the treatment to themselves in nearly all cases, unless too weak to do so.

The treatment with the quartz lamp is so simple that the patient, as we have noted, can administer it to himself. He should always be warned that, simple as it appears, it can cause painful burns if an overdose is taken, and a very painful conjunctivitis, if the eyes are not properly protected by goggles or by other means. Further than this, the patient has to accustom himself gradually to lying naked for an hour. The room, of course, should be well heated and 70° F. has been found to be a satisfactory room temperature.

The method or scheme of dosage is immaterial, provided that an erythema, if one is produced, be slight and fade quickly. If an erythema that causes slight discomfort, is produced, the treatments should be omitted for a day or two. As such intermissions delay

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the results of treatment, they should be carefully avoided by cautious increases in the dosage. Particular attention should be paid to the danger of falling asleep while under the lamp. We have recently had to treat a patient for a burn—a fourth degree erythema with vesiculation—who overexposed his whole body through falling asleep. He responded quickly to the paraffins used in the ordinary treatment of burns. To avoid such accidents an alarm clock can be obtained, which rings at the end of any desired time.

Our method is to place the lamp over the center of the area to be exposed at a distance of 36 inches (90 cm.). To accustom the patient to exposure without clothes, we expose, on the first day, only from the feet to the middle of the calves, for three minutes over the back, and three minutes over the front. The next day, with the lamp at the same distance, we expose from the feet to the middle of the thigh, the third day from the waist to the feet and the fourth day the whole body, first over the back, then over the front, each for three minutes, keeping the lamp always at 36 inches. This lamp-distance is maintained as the time of exposure is increased two minutes a day over the front and two minutes a day over the back, until the back and front are each exposed thirty minutes. It is difficult for many patients to undergo such treatment longer than one hour, and our aim has been to shorten the time of exposure when possible. Our method at this stage of treatment, is to lower the lamp from 3 feet to 1 foot from the skin, which gives us nine times the amount of light, as the amount of light any object receives is directly proportional to the square of its distance from the source of the light. To make sure that we subject the patient to no danger of burning, we divide the time of exposure, thirty minutes, by nine, and expose for three minutes only. At this close distance, we find the whole body is not exposed, and we are forced to make two exposures on the front, one with the lamp centered over the middle of the thighs, one with the lamp centered over a point midway between the ensiform cartilage and the umbilicus. Exposures should be made over corresponding points on the back, which, in fact, should be exposed first, as lying on the abdomen is very irksome to many patients. We do not increase the time of exposures for three days and then increase one minute a day over each of the four areas, until each area is exposed for ten minutes. Longer exposures can be made, but some prefer to increase the intensity of the light rather than the time of exposure. Of course, the dose of light can be increased much more rapidly in many patients, but such a scheme as outlined seldom burns any patient. Many patients burn slightly

and peel, fine flakes of skin being thrown off. At times, this is so pronounced that on undressing the whole floor is covered with fine scales. Such peeling may retard for a time the desired heliotherapeutic results. Smaller increases or maintaining the same dosage over a number of days will usually correct it. There are a few patients who, it seems, cannot take heliotherapy. Only one has fallen under our observation. She is a blonde and any dose of light, natural or artificial, that we could suggest, caused such discomfort that we had to desist. It is especially wise to protect the head, *i. e.*, to avoid exposing it to the rays in any such case. Some advocate covering also the chest from the clavicle to the nipples and the corresponding area on the back.

The results of heliotherapy depend upon many factors; the general condition of the patient, the condition and extent of disease in the intestine, the response to light treatment in general, the reaction of the skin, etc. Patients, who tan easily, are said to do better than those who do not, but our experience shows that both get results. Patients, with far advanced pulmonary and intestinal disease, with little or no remaining resistance, cannot be expected to respond to any form of stimulation, and the results obtained by heliotherapy and all other forms are, of course, usually negative. This emphasizes, however, the importance of what we have said before, viz., that all patients in the moderately advanced stage of pulmonary tuberculosis, if indeed not all in the minimal stage as well, should be subjected to a routine roentgen-ray study of the intestinal tract, in order to institute treatment in as early a stage as possible. Today, it can safely be said that, like tuberculous laryngitis, intestinal tuberculosis can be cured in many patients, even though some eventually die from their pulmonary tuberculosis. Autopsies performed by Gardner, in Saranac Lake, have confirmed this (see p. 81).

It is of interest to review briefly some results obtained by heliotherapy.

Rollier treated with natural heliotherapy 16 cases of ileo-cecal tuberculosis, between 1903 and 1913, and 9 healed, 4 improved and 2, severely affected, died. Between 1913 and 1921, he similarly treated 34 cases of intestinal tuberculosis, of which 4 healed, 15 improved, 14 remained stationary and one, with amyloid degeneration died.

Blanchet, in 1921, reported 100 cases of pulmonary tuberculosis, complicated with intestinal tuberculosis, which he had subjected to treatment with the ultraviolet ray. Of these, 25 had clinical

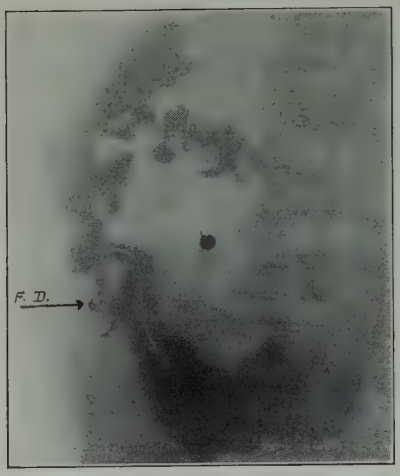

FIG. 72 (Case 5070).—First examination. Roentgen finding at six hours: filling-defect or spasm of the cecum and ceco-colon (*F.D.*) and probably areas in the hepatic flexure region. Confirmed at seven and eight hours. At twenty-four hours generalized hypermotility, the barium having been completely eliminated.

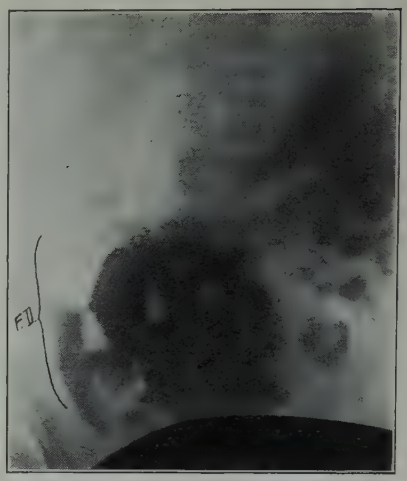

FIG. 73 (Case 5070).—Second examination, four months after the first examination. Roentgen finding at seven hours: gastric retention, filling-defect or spasm of the ascending colon (*F.D.*) more pronounced than in the first examination. (See Fig. 72); the ulceration has probably extended. Confirmed at six and eight hours. At twenty-four hours generalized hypermotility, all the barium having been completely eliminated.

FIG. 74 (Case 5070).—Third examination, seventeen months after the first examination. Roentgen finding at eight hours: filling-defect (*F.D.*) of much of the proximal colon; degree of irritability seems to be less as there was no spasm, which was seen on the first examination. At twenty-four hours generalized hypermotility, all the barium having been completely eliminated. Note the half-moon effect at the ileo-cecal sphincter (*X*).

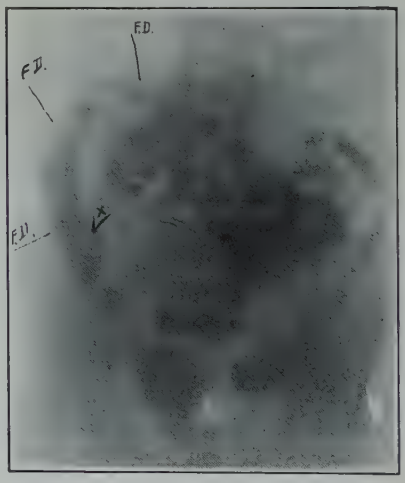

FIG. 75 (Case 5070).—Fourth examination twenty-five months after the first examination. Roentgen finding at seven hours: filling-defect of the proximal colon (*F.D.*), generalized hypermotility (head of the column in the rectum); note the persistence of the half-moon effect at the ileo-cecal sphincter (*X*) as seen in Fig. 74.

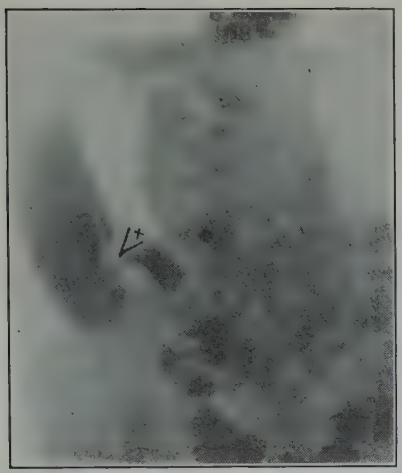

FIG. 76 (Case 5070).—Fourth examination. Roentgen finding at eight hours, confirming seven-hour observation; note at this time the half-moon effect at the ileo-cecal sphincter (*X*) is more decided. At twenty-five hours generalized hypermotility, all the barium having been completely eliminated.

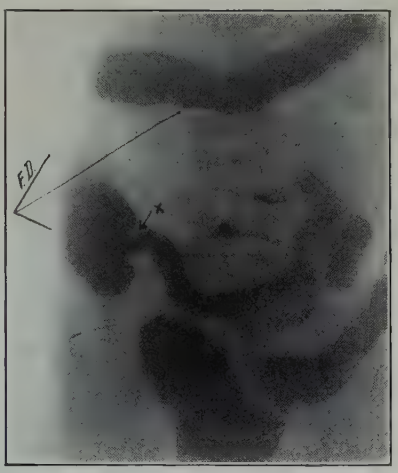

FIG. 77 (Case 5070).—Fourth examination. Roentgen finding after barium enema: lack of haustration in the proximal colon (*F.D.*); half-moon effect at the sphincter (*X*); incompetent ileo-cecal valve.

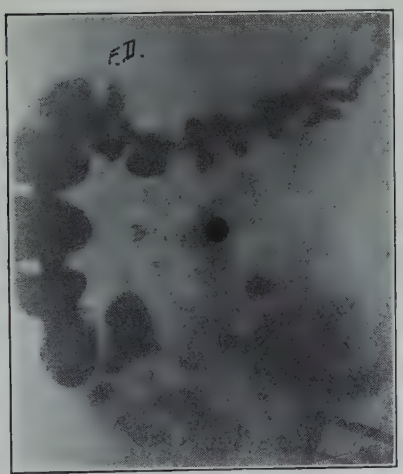

FIG. 78 (Case 5070).—Fifth examination thirty-five months after the first examination. Roentgen finding at nine hours: the irritability decidedly less; reappearance of haustra (however the haustra are very deep and appear spastic); probable filling-defect or spasm (*F.D.*) at the hepatic flexure. Confirmed at seven and eight hours. At twenty-four hours there is still generalized hypermotility, all the barium having been completely eliminated.

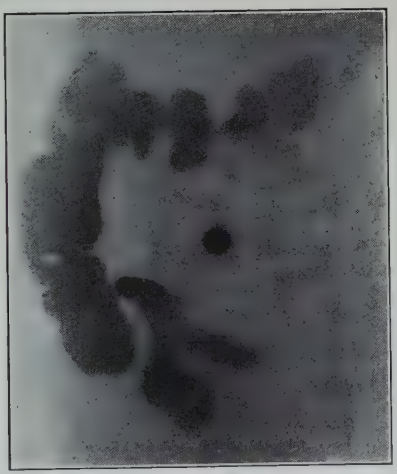

FIG. 79 (Case 5070).—Sixth examination forty-six months after the first examination. Roentgen finding at eight hours: very little evidence of filling-defect, though there is some irregularity in the outline of the cecum. Confirmed at seven and nine hours. At twenty-four hours there is generalized hypermotility, all the barium having been completely eliminated.

symptoms of intestinal tuberculosis and, after treatment, 10 lost all symptoms and the roentgen-ray picture was normal, 5 were better, 6 failed and 4 died. Of 50 active cases, 13 improved, 14 received some help, 10 remained the same, 8 failed and 5 died. Of 50 cases more or less quiescent, but at a standstill, 15 improved considerably, 15 improved slightly, 15 remained the same and

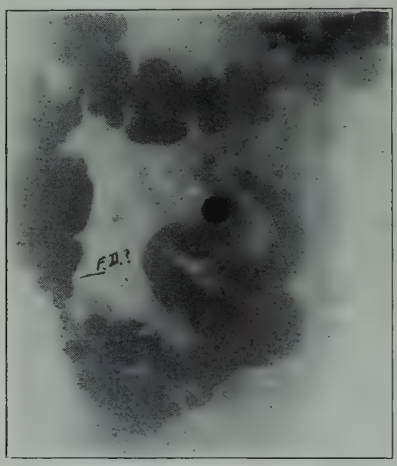

FIG. 80 (Case 5070).—Seventh examination fifty-two months after the first examination. Roentgen finding at nine hours: irregularity or indefinite filling-defect (*F.D.*?) at the cecum. Somewhat confirmed at seven and eight hours. At twenty-four generalized hypermotility, all the barium having been completely eliminated.

Note.—The generalized hypermotility observed at twenty-four hours in each of the seven examinations is very significant and particularly so in the later examinations, at which time there was very little filling-defect and no spasm.

Clinical Finding.—Male, single, aged twenty-two years, with moderately advanced pulmonary tuberculosis. Intestinal tuberculosis discovered on routine roentgen-ray examination. Fistula-in-ano. No treatment for six months. Worse. Severe diarrhea and pain. Roentgen-ray treatment for four months. Improved. Ultraviolet-ray treatment and marked improvement. Now occasional soft movement.

5 failed. Of the whole number 28 showed good continued improvement, 29 some gain, 25 remained stationary, 13 failed and 5 died. Many of these patients were still under treatment when the paper was written, but those, who recovered, were still well two years later.

Stewart, of Ninette, subjected 77 patients, many with gross pulmonary and intestinal lesions, to treatment by ultraviolet ray for

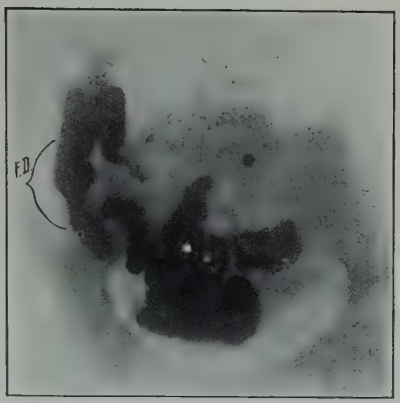

FIG. 81 (Case 129).—Roentgen finding at six hours: filling-defect (*F.D.*) of the ascending colon; absence of spasm indicating a chronic ulcerative process (compare with Case 112). Confirmed at seven and eight hours. At twenty-four hours generalized hypermotility, all the barium having been eliminated.

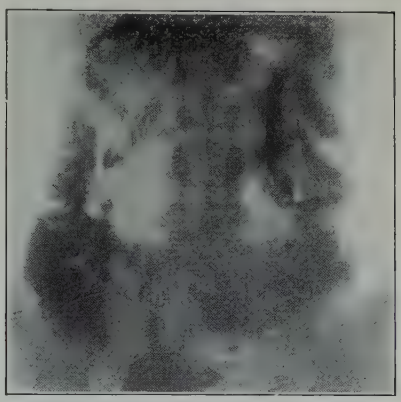

FIG. 82 (Case 129). — Thirty-two months after first examination. Roentgen finding at seven hours: gastric retention; reappearance of haustra; no evidence indicating ulceration.

FIG. 83 (Case 129).—Roentgen finding at eight hours, confirming seven-hour examination.

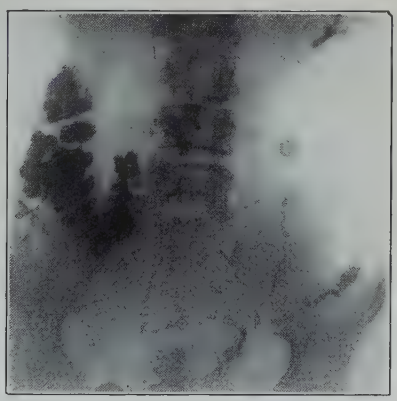

FIG. 84 (Case 129).—Roentgen finding at twenty-four hours: average normal colonic retention; disappearance of generalized hypermotility.

Autopsy Finding.—See Case 129. Table XXXVI.

Clinical Finding.—Female, single, aged twenty-six years. Pulmonary tuberculosis began 1919. In 1920, infiltration right lung. Abdominal symptoms, pain and diarrhea. Tuberculous colitis diagnosed prior to abdominal symptoms. Ultraviolet-ray treatment for ten months with relief of symptoms. Later, artificial pneumothorax. In 1923, died from pneumonia in opposite lung which was not of pyogenic origin. (Gardner).

FIG. 85 (Case 109).—First examination. Roentgen finding at six hours: gastric retention; generalized hypermotility (head of the column in the rectum); filling-defect or spasm of the proximal colon (*F.D.*). At the twenty-fourth hour generalized hypermotility, all the barium having been eliminated.

FIG. 86 (Case 109). — First examination. Roentgen finding after barium enema: spasm of the ascending colon; also ragged (furred) appearance of the first half of the transverse colon (*F.D.*) confirming the "meal" examination.

FIG. 87 (Case 109).—Second examination, nineteen months after first examination. Roentgen finding at seven hours: filling-defect (*F.D.*) of the cecum. Confirmed at six and eight hours. Note that there is less localized irritability than in the previous examination. However, there is still generalized hypermotility, the bowel emptying in twenty-four hours.

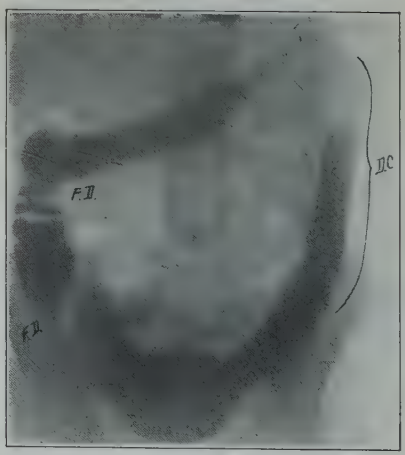

FIG. 88 (Case 109).—Second examination. Roentgen finding after enema: filling-defect (*F.D.*) of the cecum and hepatic flexure region; non-haustration (tube-like effect) of the distal colon (*D.C.*).

a considerable time, and found two-thirds showed improvement. Of 17 cases with gross active lung lesions and acute active intestinal disease, 1 improved temporarily, 2 more permanently (1 operated upon) and 14 did not improve. Of 10 with gross active pulmonary lesions and less active intestinal disease, 2 improved temporarily and 8 did not improve. Of 3 who had pulmonary disease less gross and active, but whose intestinal disease was active, all improved, 2 after operation. Of the remaining 47, whose pulmonary disease

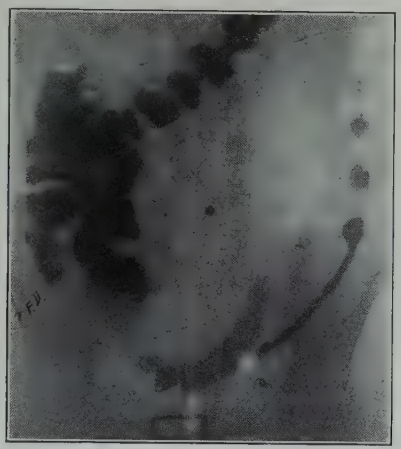

FIG. 89 (Case 109).—Third examination thirty months after first examination. Roentgen finding at seven hours: slight filling-defect (*F.D.?*) at the cecum; generalized hypermotility (head of the column in the sigmoid). Confirmed at six and eight hours. At twenty-four hours the barium had been completely eliminated.

Operative Finding.—See Case 109. Table XXXVI.

Clinical Finding.—Male, married, aged thirty years. Pulmonary tuberculosis began December, 1915 and slowly advanced. In August, 1917, infiltration of entire right lung with almost consolidation in upper one-third. In October occasional loose bowels. No marked diarrhea. Intestinal tuberculosis diagnosed, October 22, 1919. Operation, appendectomy. Gradual recovery from intestinal and pulmonary tuberculosis.

was not so gross or active and whose intestinal disease was not more than moderately active, all but 5 made what seemed to be apparently permanent improvement or recovery, one was improved temporarily and 4 failed to improve.

Reh, who advocated heliotherapy and roentgen-ray treatment of all slight cases of intestinal tuberculosis (*i. e.*, those with diarrhea as the single outspoken symptom), and surgical treatment for those with loss of strength, high fever or uncontrollable diarrhea, has

reported a case, a nervous woman, married, aged thirty-six years, in which on account of symptoms a fixation of the uterus was done. At operation, tuberculosis of the cecum and ileum was found accom-

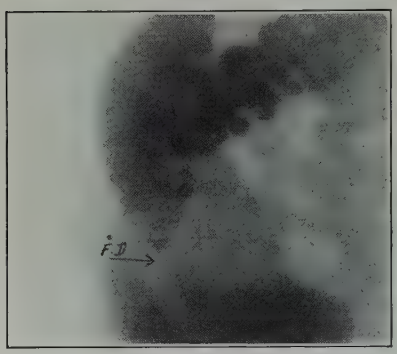

Fig. 90 (Case 5238).—Roentgen finding at seven hours: filling-defect or spasm (*F.D.*) of the cecum; generalized hypermotility (head of the column in the sigmoid).

Fig. 91 (Case 5238).—Roentgen finding at eight hours: filling-defect (*F.D.*) of the cecum still present.

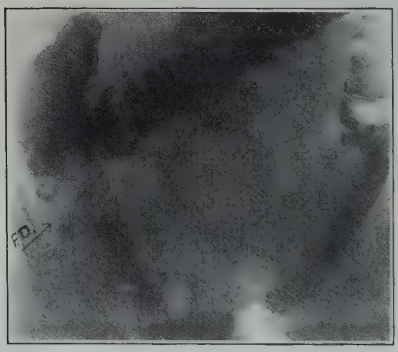

Fig. 92 (Case 5238).—Roentgen finding at nine hours: filling-defect (*F.D.*) of the cecum still exists; note that the ileum has emptied considerably since the seventh hour. Roentgen finding at twenty-four hours: generalized hypermotility, all the barium having been eliminated.

Fig. 93 (Case 5238).—Roentgen finding following barium enema: indefinite filling-defect (*F.D.*) of the cecum; note that this is decidedly less marked than the filling-defect seen from seventh to ninth hour following the barium meal.

panied by frequent adhesions, which he freed, and after washing out the abdomen with hot salt solution, closed it. Deep roentgen-ray therapy was given (6 treatments of ten minutes each over four

fields in a period of three months). She was subjected then to heliotherapy and the hygienic-dietetic treatment, and made remarkable progress, but after five months the hysterical symptoms returned with pain in the right lower quadrant and some diarrhea. A second operation revealed adhesions and signs of what he interpreted as healed intestinal tuberculosis. The complete recovery was uneventful.

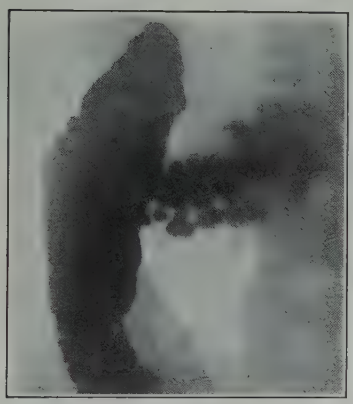

FIG. 94 (Case 5238).—Seven months after first examination. Roentgen finding at nine hours: practically complete disappearance of the previous filling-defect or spasm. Confirmed at seven and eight hours.

FIG. 95 (Case 5238).—Roentgen finding at twenty-four hours: average normal colonic retention and outline.

Clinical Finding.—Male, married, aged thirty years. Pulmonary tuberculosis began in the autumn of 1918. In June, 1919, infiltration right apex and left upper one-half. Tuberculous colitis diagnosed, April, 1920. Ultraviolet-ray treatment, good results. Well four years later.

Mention has already been made of the admirable study of the symptoms of intestinal tuberculosis by Erickson. At the same time, he made a study of the changes in the symptoms of 81 positive cases, which had been subjected to ultraviolet-ray treatment. Of these, 69 (85.2 per cent) had a favorable result; in 24.7 per cent, the symptoms were entirely relieved; in 47 per cent, they were much improved; and in 13.5 per cent, they were slightly but definitely relieved. In 12 (14.8 per cent), there was no benefit but almost all of these patients were very sick, often hopelessly ill with pulmonary tuberculosis. In fact, they were cases that would give little hope of improvement under any conditions.

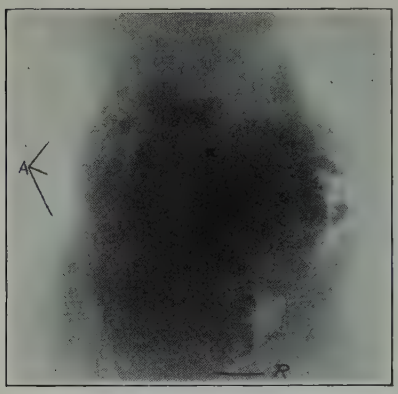

FIG. 96 (Case 17107).—Roentgen finding at nine hours:[1] small amount of gastric retention (head of the column in the rectum (*R*); ileum fairly well filled; note the ascending colon is empty (*A*). The above findings were definitely seen fluoroscopically. Confirmed at ten and eleven hours.

[1] Examination started at nine hours due to the fact that very little barium could be seen in the colon at earlier hours).

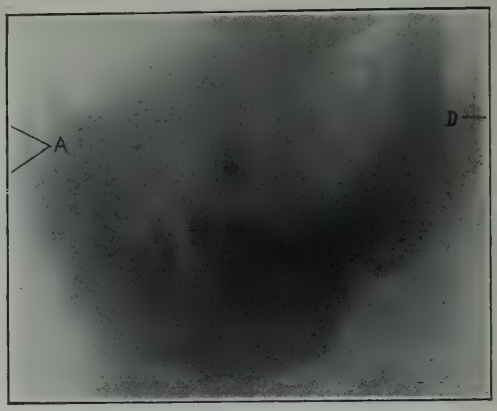

FIG. 97 (Case 17107).—Roentgen finding at twelve hours: the descending colon (*D*) is well filled; ascending colon (*A*) is empty; barium still remains in the ileum. At twenty-four hours, generalized hypermotility, all barium having been eliminated. Note that there is hypermotility at twenty-four hours in spite of the delay in entering the colon at nine hours.

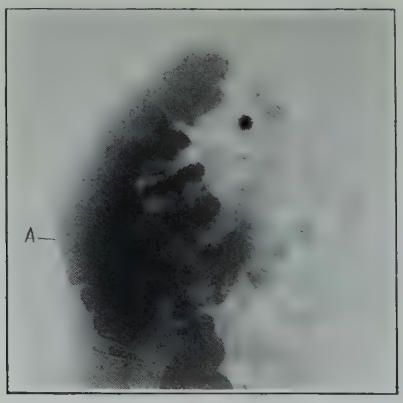

FIG. 98 (Case 17107). — Thirty-six months later. Roentgen finding at seven hours: note the average normal motility and the smooth haustration. Confirmed at eight, nine and twenty-four hours.

Clinical Finding.—Male, married, aged twenty-seven years. Pulmonary tuberculosis began June, 1920. July, 1920, infiltration right lung. Abdominal symptoms, December, 1920, gas, nausea, vomiting, alternating constipation and diarrhea. Ultraviolet-ray treatment one year. Symptomatically, complete recovery. February, 1925, well.

It is of interest to see how soon definite results appeared after the treatment was instituted. Definite improvement occurred,

	Per cent.
In one month in 37 cases	45.7
In two months in 13 cases	16.0
In three months in 13 cases	16.0
In more than three months in 6 cases	7.5

Thus 61.7 per cent were relieved inside of two, 74.7 inside of three and all but 2 cases inside of six months.

In his study Erickson separated the symptoms and grouped them as follows:

TABLE XLIV.

	Entirely relieved.		Much improved.		Slight improvement.		Not improved.	
		%		%		%		%
Pain	30	60	9	18	5	10	6	12
Constipation	5	42	3	25	1	8	3	25
Diarrhea	19	40	21	44	4	8	4	8
Nausea and vomiting	26	59	8	36	5	25	5	2.5
General digestive disturbance	11	28	18	46	7	18	3	7.0
General systemic disturbance	7	44	9	56	0		0	

Thus, it would appear that pain, nausea and vomiting are most quickly relieved by ultraviolet-ray treatment, but diarrhea, general digestive disturbances, and general systemic disturbances are relieved or greatly benefited. Constipation is less affected but is aided.

These facts are presented graphically in Fig. 99. In this chart, the total length of time that the symptoms were present before the lamp was used, the length of time that the lamp was used until favorable results were obtained, and the length of time that the patients have been relieved of severe symptoms, are compared. It must be taken into consideration that the relief of symptoms in the last-named group will as time goes on become of longer and longer duration. Even as it stands it is highly suggestive.

In another chart (Fig. 100) is shown the relation of the duration of symptoms, previous to treatment with ultraviolet light, to the duration of such symptoms after this treatment was begun. In all, 69 cases were studied, as the following figures on page 244 show:

TABLE XLV.

Duration of symptoms.	No. of cases.	Definitely improved in			
		1 month.	2 months.	3 months.	Over 3 months.
Less than two months	11	5	2	3	1
Two to four months	26	13	5	5	3
Five to nine months	16	12	1	2	1
Nine to twelve months	8	4	2	2	0
Over twelve months	8	3	3	1	1
	69	37	13	13	6

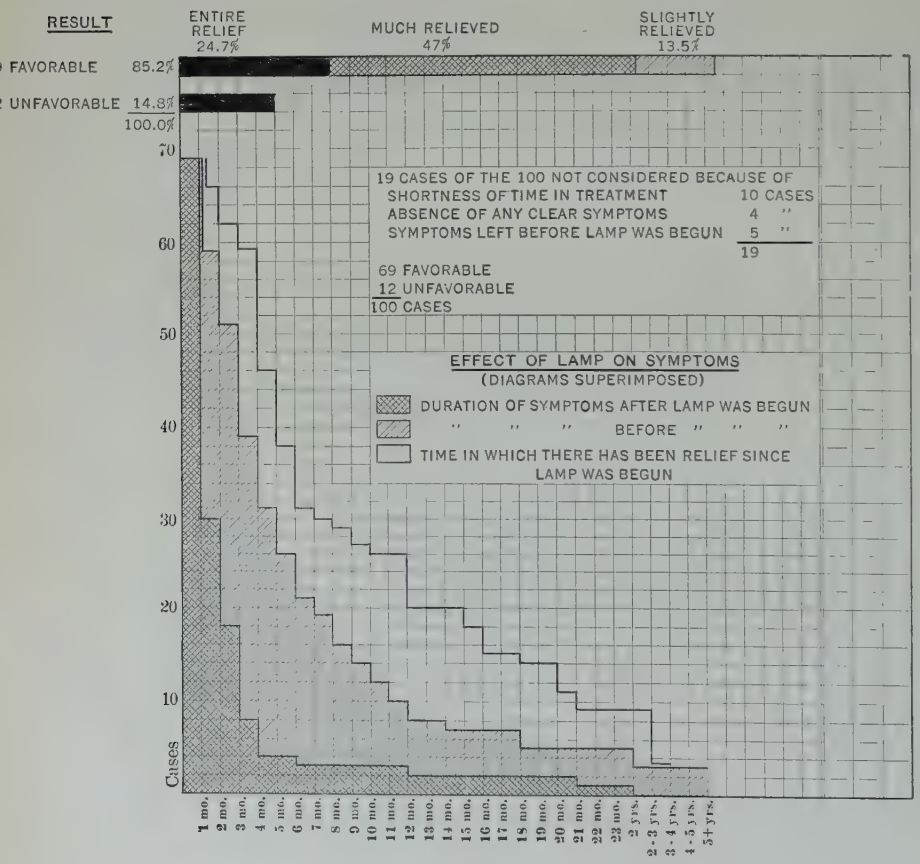

FIG. 99.

Such figures as these bear out Erickson's view, that the results of the ultraviolet-ray treatment are more than coincidental and are not due to the fact that in intestinal tuberculosis the symptoms run a naturally limited course.

If we consider only those cases in which the symptoms have persisted over two months, eliminating in this way all cases in which the symptoms have not had time to become firmly established, we find that of the 58 cases, 55 per cent were improved in one month, 19 per cent within two months and 17 per cent within three months. Thus within three months 91 per cent of all cases were improved. However, when all the cases are considered the figures are practically the same.

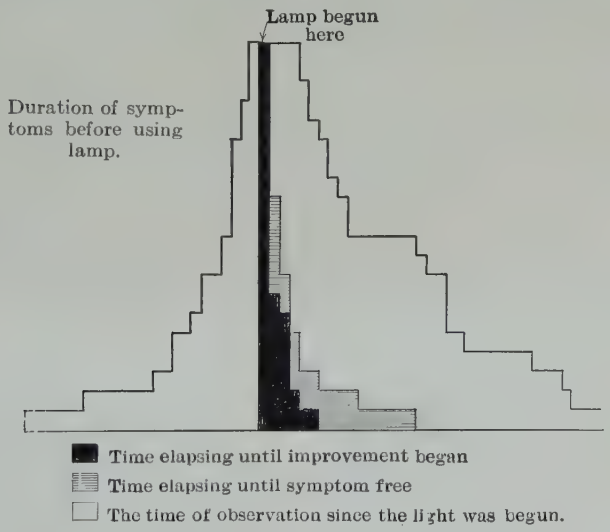
Lamp begun here
Duration of symptoms before using lamp.
Time elapsing until improvement began
Time elapsing until symptom free
The time of observation since the light was begun.

FIG. 100.

A study of 69 cases, that received complete relief of their symptoms, is shown in Fig. 100. The duration of symptoms before the ultraviolet-ray treatment was begun, the lapse of time after the onset of treatment until improvement was noted, the lapse of time until the patients were symptom-free, and the length of time that they have been under observation since losing all symptoms, are compared. It will be noted that, after treatment for one month, 65 per cent were improved and 40 per cent were free of symptoms, and after two months, the figures were 70 per cent and 60 per cent respectively. After treatment for six months, all were improved and 85 per cent had lost all intestinal symptoms. All patients

were observed for at least three months, 50 per cent for sixteen months, and 20 per cent for two years or longer after cessation of treatment.

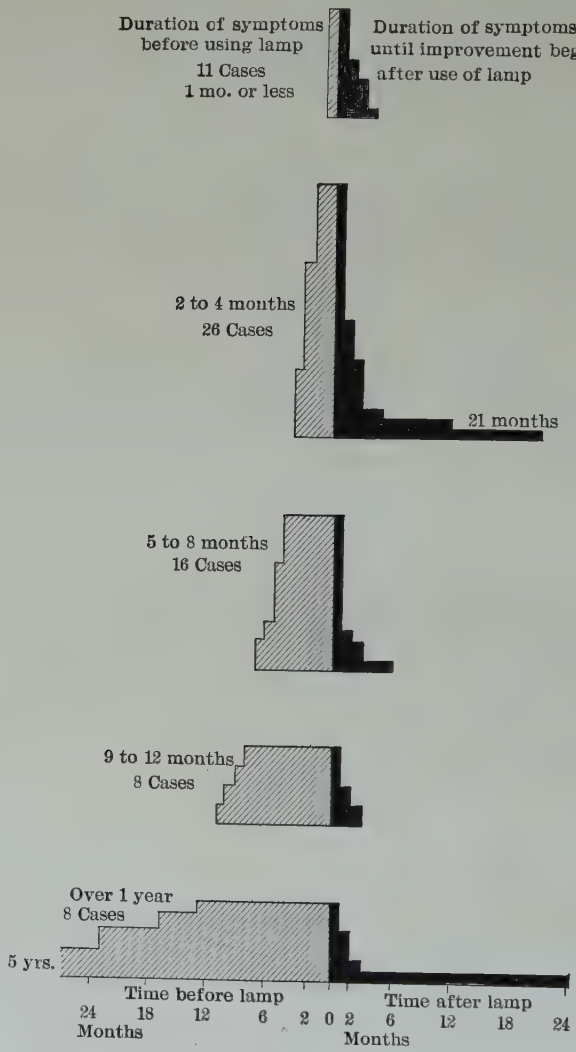

FIG. 101.—Showing relative duration of symptoms before lamp, to time improvement began in 69 cases having had symptoms before using lamp.

The length of the duration of symptoms, before and after taking the ultraviolet-ray treatment, is graphically presented in each group. This is done, because it may seem from the previous charts and

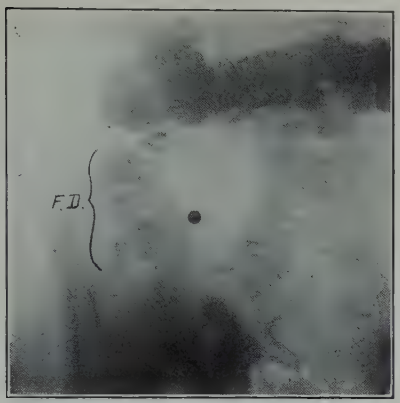

FIG. 102 (Case 5135).—Roentgen finding at five hours: general hypermotility (head of the column in the sigmoid), filling-defect and spasm of the ascending colon (*F.D.*). Confirmed at four, six and seven hours. At twenty-four hours the barium had been completely eliminated.

FIG. 103 (Case 5135).—Roentgen finding after enema: filling-defect of the cecum (*F.D.*). Five minutes after the administration of the barium enema there was a spasm of the colon from the cecum to the descending colon (see Fig. 104). Does this indicate involvement of all this area? Unlikely.

FIG. 104 (Case 5135).—Roentgen finding: spasm or contraction of the ascending, transverse, and descending colon (see Fig. 103).

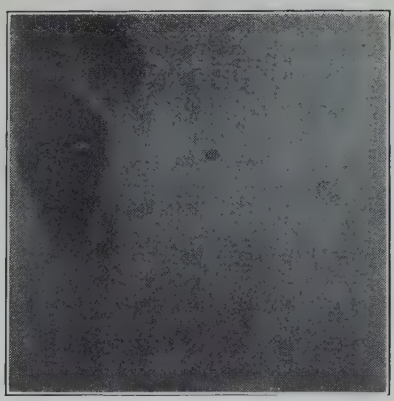

FIG. 105 (Case 5135).—Second examination, ten months after first examination. Roentgen finding at six hours: gastric retention; practically complete disappearance of the previous filling-defect or spasm of the cecum. At twenty-four hours colon fairly well filled throughout.

Clinical Finding.—Male, single, aged twenty-seven years. Pulmonary tuberculosis began September, 1918. Infiltration right lung and left upper half. Pulmonary condition favorable. Bowels constipated, never diarrhea. Tuberculous colitis diagnosed, March, 1919. Ultraviolet-ray treatment with excellent results. Classified chronic four years later.

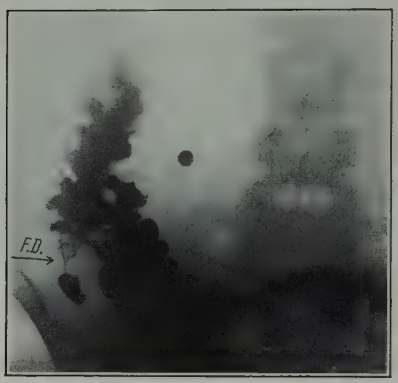

FIG. 106 (Case 4655).—Roentgen finding at seven hours: filling-defect of the cecum (*F.D.*).

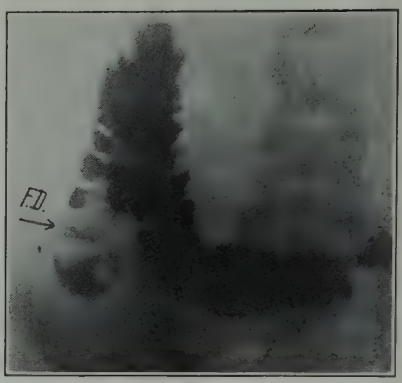

FIG. 107 (Case 4655).—Roentgen finding at eight hours: spasm or filling-defect of the cecum (*F.D.*). Confirmed at seven hours. At twenty-four hours generalized hypermotility, all the barium having been eliminated.

FIG. 108 (Case 4655). — Fourteen months after first examination. Roentgen finding at seven hours: practically complete disappearance of previous filling-defect or spasm of the cecum (*C*). Confirmed at eight and nine hours.

Clinical Finding.—Female, single, aged twenty-four years. Pulmonary tuberculosis began December, 1916. Relapsed, 1920. Infiltration right and left upper lobes. Tuberculous colitis diagnosed, March, 1921. No gastro-intestinal symptoms. Later alternate diarrhea and constipation. Much benefit from ultraviolet-ray treatment. Three years later relapsed and died.

FIG. 109 (Case 14769).—First examination. Roentgen finding at seven hours: filling-defect of the cecum and ceco-colon (*F.D.*)—this finding not marked.

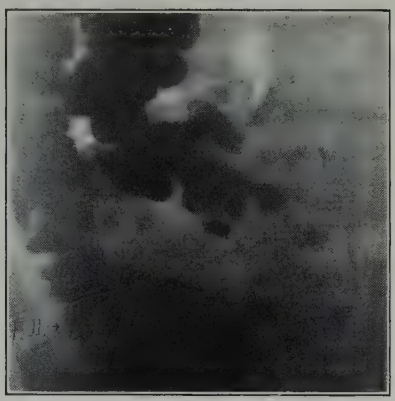

FIG. 110 (Case 14769).—First examination. Roentgen finding at nine hours: filling-defect of cecum (*F.D.*). Confirmed at seven and eight hours. Roentgen finding at twenty-four hours: barium completely eliminated.

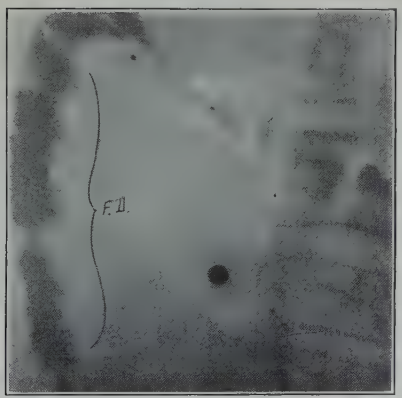

FIG. 111 (Case 14769).—Second examination, four months after the first examination. Roentgen finding at nine hours: filling-defect or spasm of the ascending colon and hepatic flexure region (*F.D.*). Confirmed at seven and eight hours. Roentgen finding at twenty-four hours: barium completely eliminated.

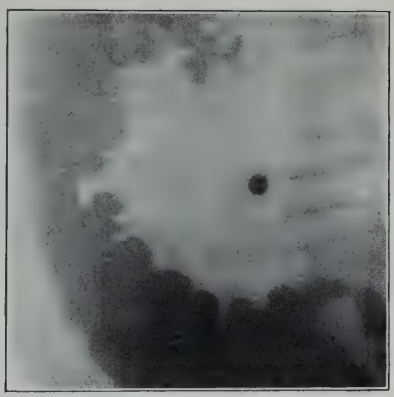

FIG. 112 (Case 14769).—Third examination twelve months after the first examination. Roentgen finding at nine hours: no definite spasm or filling-defects, though the ascending colon is somewhat irregular. This examination practically negative, confirmed at seven and eight hours.

Clinical Finding.—Female, married, aged thirty-six years. June, 1920, right lung, infiltration upper third with questionable cavity. Left lung, upper half. Abdominal symptoms began November, 1920. Tuberculous colitis diagnosed, March, 1921. Ultraviolet-ray treatment, April, 1921 and continued for two years. All symptoms disappeared. Present condition, absolutely well.

figures that the symptoms may have a certain natural duration, and that the apparently good results shown by those undergoing ultraviolet-ray treatment, are due to the fact that the disease is in a certain degree self-limited. A study of these charts will show that good results are obtained in each of the three groups. Perhaps results are more striking in the cases that have a duration of from eight to twelve months. It may be said that this is so, because the disease has in these cases run its course. The difference, however, is so slight in comparison to the uniformly good results in all the groups that we can hardly avoid the conclusion, that we have in the quartz mercury-vapor lamp a therapeutic agent that is of great value. So striking are these results, that we may almost put the burden of proof on those who think the results are merely coincidental.

While the results of the ultraviolet-ray treatment have been graphically shown by Erickson's charts, it seems wise to record the results in 180 cases treated in Saranac Lake Village, some of which are included in his figures. Of the 209 cases studied, 29 received no ultraviolet-ray treatment, and 5 (17 per cent) are living and 24 (83 per cent) are dead. Of the 180 treated cases, 116 (65 per cent) are living and 35 per cent dead.

	Living.	Dead.
With ultraviolet-ray treatment . . .	116 (65%)	64 (35%)
Without ultraviolet-ray treatment . .	5 (17%)	24 (83%)

If we separate the cases into minimal, moderately, and far advanced stages we get the following figures:

TABLE XLVI.

	Ultraviolet-ray treatment.		No ultraviolet-ray treatment.	
	Living.	Dead.	Living.	Dead.
Minimal	2	0	0	0
Moderately advanced . .	63 (77%)	19 (23%)	1 (8%)	11 (92%)
Far advanced	51 (53%)	45 (47%)	4 (24%)	13 (76%)
	116	64	5	24

While there can be no question about a classification into living and dead, it may fail to tell the whole story and, consequently, it was decided to divide the cases treated with the lamp into six

groups, *i. e.*, those with no results, with questionable results, with slight, considerable and marked improvement and cases treated less than a month.

TABLE XLVII.

			No results.	?	+	++	+++	Less than 1 month.
Living 116 (65%)	Minimal	2	..	..	..	..	2	
	Moderately advanced	63	..	4	17	12	28	2
	Far advanced	51	1	8	16	11	15	
Dead 64 (35%)	Moderately advanced	19	2	2	4(*b*)	2	6(*c*)	3(*a*)
	Far advanced	45	7	10	12	8	5(*d*)	3
		180	10	24	49	33	56	8

(*a*) One had amœbic dysentery as well.
(*b*) One had cardiac disease.
(*c*) One had fatal hemoptysis.
One had relapse after excellent results (treatment stopped).
One had hemoptysis and meningitis.
One had intestinal relapse.
One had ruptured lung.
(*d*) One had meningitis.

The following table of percentages based on Table XLVII is of interest:

TABLE XLVIII.

	All cases.		Moderately advanced		Far advanced.	
	Improved.	Much improved.	Improved.	Much improved.	Improved.	Much improved.
Of whole number . . .	77	30	84	40	70	21
Of dead	59	16	60	30	58	9
Of living	87	39	92	44	82	31

This seems to indicate that a large majority of all the cases are more or less benefited by the general ultraviolet-ray treatment but that the ultimate results depend not alone upon the intestinal lesions but also upon the state of the lungs. In 26 of these cases, the roentgen-ray examination after treatment showed no longer any evidence of pathological change. Gardner's work has also shown that in the intestines tuberculous lesions may retrogress as may similar lesions in the larynx, even though the pulmonary condition advances to death.

A similar study of the 106 cases treated at the Trudeau Sanatorium for intestinal and pulmonary tuberculosis gives the following figures. It must be borne in mind that these cases are often of a less severe type than those treated in Saranac Lake Village.

TABLE XLIX.

	Living.	Dead.
With ultraviolet-ray treatment	76 (88%)	10 (12%)
Without ultraviolet-ray treatment . . .	5 (25%)	15 (75%)

From a more detailed study, dividing the cases into those who received no benefit, questionable results, slight, considerable and marked improvement, we obtain the following table.

TABLE L.

		No. of cases.	No results.	?	+	++	+++	Less than one month.
Living	Minimal	0						
	Moderately advanced	55	5	0	6	19	16	9
	Far advanced	21	5	0	4	5	5	2
Dead	Minimal	0						
	Moderately advanced	5	1	0	2	0	1	1
	Far advanced	5	1	0	1	1	2	0
Total		86	12	0	13	25	24	12

If we deduct those who received the lamp less than one month and consider the remaining 74 patients, we find 16 per cent received no benefit and 65 per cent were considerably benefited, some of whom were apparently healed. The roentgen-ray examination revealed that 22 (31 per cent) of these cases no longer showed any pathological changes.

Unquestionably more of the 869 positive cases, many of which improved markedly symptomatically, would have been negative on roentgen-ray examination, had it been performed. As a matter of fact, of those cases which had a subsequent examination 85 had become negative.

Johannes Mueller, in the *Handbuch der Tuberkulose*, makes no mention of heliotherapy under the head of the treatment of intestinal tuberculosis. Rollier, of course, has long advocated natural heliotherapy in intestinal tuberculosis.

ROENTGEN-RAY TREATMENT.

Several years ago, the cost of ultraviolet-ray treatment was so high that we were forced to cast about for other and cheaper forms of radiant-energy treatment. We found that we could give roentgen ray-treatment, free of all charge, to some poor patients, who had hardly enough funds for ordinary maintenance. Accordingly in 1919, we began roentgenologic treatment and used as an average dose 5 milliampère minutes (90 kilo-volts, 2.5 to 3 milliampères, filtered through 3 mm. of aluminum, with the tube at a distance of 35 cm.). The results were in some cases very gratifying. We quickly found out that the intestines are very susceptible to roentgen-rays and that reactions of some intensity, with recurrence or increase of all the intestinal symptoms, could easily be brought about. We ordered our dosage accordingly, and now combine roentgen-ray treatment with ultraviolet-ray therapy in many cases, where ultraviolet-ray therapy alone does not give us the desired results. Bacmeister, in 1921, claimed precedence for this form of treatment. Once again we would like to recall and to warn against the danger of the cumulative effect of roentgen-rays. Warren and Whipple have emphasized this and called attention to their experimental work, which shows that the mucosa of the small intestine is very susceptible to hard and short-wave roentgen-rays. An overdose may produce intestinal ulceration, which we are trying to heal. A diffuse exposure of 240 to 400 milliampère minutes would cause fatal intoxication according to the authors mentioned, as some out-patients reacted to from 5 to 10 milliampère minutes.

Glaser (in 1924) has reported a case of severe colitis, following roentgen-ray treatment, and reviewed some work of others. Franz, in 1917, reported to the Berlin Medical Society a case of death from severe intoxication after roentgen-ray treatment, due to ulcerative enteritis. He found the stomach secretion in the healthy uninfluenced. Schmidt, in 7000 cases, produced no harm. In 1920, Fischer (Frankfurt) reported 3 cases of severe intestinal necrosis after deep ray therapy. Heck, in 1921, had a similar case. Lieck, in 1922, says many roentgen-ray intestinal injuries occur and reported a case of a married woman, who was curetted for menorrhagia and then treated with roentgen-ray. After the fourth treatment, severe diarrhea with mucus, blood and pus occurred. The symptoms continued for weeks. An examination showed severe ulcerative colitis, seen with the proctoscope. In spite of the most careful treatment, the disease advanced, and "now after a half-year of internal treatment an operation must be performed."

THE INTRAPERITONEAL INJECTION OF OXYGEN.

The beneficial effects of simple laparotomy upon certain types of tuberculous peritonitis have, of course, suggested its use in intestinal tuberculosis, but, on the whole, it has seemed to exert little if any effect. For some years, the inflation of oxygen into the tuberculous peritoneum has been advocated (Bainbridge, Rost, Purves and Bilcliffe, etc.) and excellent results reported. The recent use of pneumo-peritoneum in the diagnosis of certain abdominal conditions has familiarized many with the method. Accordingly, it is not strange that intraperitoneal injections of oxygen have been used in the treatment of intestinal tuberculosis. Jelks has recently (1923) written of this treatment, which has been followed by improvement and cure, quoting 3 cases of J. A. Price.

Laney injected oxygen by this method in 3 patients, 2 almost moribund, who died, and 1 case in which ultraviolet-ray treatment on the trunk, from September to February, had failed, was benefited and was alive at the end of a year. He used the same technic as in artificial pneumothorax, punctured the abdominal wall over McBurney's point, injected 300 cc. and turned the patient on his right side to keep the gas in. In eleven days he gave 1500 cc.

The technic has been well described by E. W. Hayes. Either air (80 per cent oxygen) or pure oxygen, which is better, can be used. The procedure closely approximates that employed for artificial pneumothorax. The usual site of injection is $1\frac{1}{2}$ inches to the right and the same distance below the umbilicus. The abdominal wall, especially the peritoneum, is well anesthesized with novocain containing a little adrenalin. A needle similar to the one used for collapse therapy is then introduced into the peritoneum, or a Salimen cannula has been recommended by some. Any apparatus satisfactory for artificial pneumothorax may be used. The pressure is at first neutral and no oscillations are noted. After the injection of 200 to 300 cc., the pressure will often rise to + 2 to 4 cm. (water) and oscillations of 1 cm. may be noted. At this time liver dulness also begins to disappear. It is wiser to inject only 300 to 500 cc. at first, though 1200 cc. in the absence of adhesions may cause no further rise of pressure. Such an amount, however, may bring about gas-pains or a blown-up feeling. Depending upon the condition of the patient, the intervals between injections vary from one to three weeks. Only gentle pressure should be used during the injection.

SURGICAL TREATMENT.

An attempt to discover the earliest lesions of intestinal tuberculosis, so as to be able to select cases suitable for surgical treatment, led us, as we have already stated, to make this study of intestinal tuberculosis. We realized that patients with pulmonary tuberculosis were poor surgical risks, and that, when this disease was complicated with intestinal tuberculosis, the risks were often almost desperate, but, without some intervention, our patients slowly but surely died. In the surgical treatment of the following cases we have depended chiefly upon Prof. Edward Archibald, of McGill University, who operated upon most of our cases and developed the various methods of surgical attack to which we have subjected our patients.

Inasmuch as intestinal tuberculosis usually begins in the ileocecal region, we hoped, that by discovering it early enough in its evolution, that we could excise the whole process. If this were impossible, we hoped that short-circuiting the bowel and excluding the diseased area would give rest to the diseased area, much relief symptomatically, and lead, eventually, to recovery.

In 1907, Conrad Brunner published in the *Deutsche Chirurgie,* a study of tuberculosis, actinomycosis and syphilis of the stomach and intestine. The section on intestinal tuberculosis is devoted largely to its surgical treatment and is, as far as we know, the best study of the whole problem. He advocates submitting to operation those with:

1. Localized intestinal tuberculosis, often of years' duration, leading to stenosis or phlegmonous forms; the hyperplastic form of cecal tuberculosis (this was the starting point of surgical treatment of intestinal tuberculosis); abscesses, either for palliative or curative purposes; and tuberculous appendicitis.

2. Stenosis—if the general condition is good. If advanced pulmonary tuberculosis, or double renal tuberculosis, or nephritis is present, one may perform a laparotomy, but internal treatment is better.

3. Perforation—if the patient can stand the operation. Fecal abscess and fecal fistula are to be treated surgically.

4. Certain doubtful risks—in disseminated tuberculous ulcers without stenosis, avoid operation if the pulmonary condition is advanced with cavities. If localized ulcers are present and can be removed, and the other organs and the general condition are good,

operation may relieve, but healing may occur without operation and so surgical treatment is of doubtful value.

Brunner has collected many cases and reports his own as well. In 125 cases of total resection for cecal tuberculosis, the operative mortality varied from nothing in 5 cases to 50 per cent in 6. In all, the operative mortality was 25 per cent, and 21 died later (7 from intestinal tuberculosis, 6 from pulmonary tuberculosis). In 7 cases of partial resection of the cecum, 6 healed.

For exclusion or short-circuiting of the intestine, Maissonneuve performed the first operation and Billroth the first outside of France. Lateral anastomosis was done in 36 cases, which of course afforded no sure proof of the diagnosis. The operative mortality was 8.3 per cent (3 cases). The later state of 10 showed 8 died (cause unknown, 3; tuberculosis, 5). The results were more than palliative for 7, 6 of whom were improved and lived over one year; 3, all improved, lived over two years; 1, three years, 1, six years but died from pulmonary tuberculosis; 2, both well, lived seven years. Seven cases had fecal fistula. Many cases of the hypertrophic type, in which pulmonary tuberculosis is uncommon, were included in the whole group.

Unilateral exclusion, first performed by Hacken in 1861, was performed in 7 cases; in 2, only palliative results followed and 5 died. In 23 cases of total bilateral exclusion, 17 had one end placed in the wound and 4 were closed and developed fistula. The operative mortality was 8 per cent (2 cases), and later 3 died, 1 each from pneumonia, pulmonary tuberculosis, and diarrhea and marasmus. Of 10 traced cases, 5 were alive and better after one year, 2 after two years, 2 after three years and 1 after six years. This, Brunner believes, is the best operation, with which Archibald agrees, if both ends of the excluded gut are placed in the abdominal wall. Simple laparotomy, Brunner states, has helped a few cases. He gives in great detail the results of Campiche's study of 154 cases, of which one-third had pulmonary tuberculosis. The results were practically the same as in Brunner's series. It must always be borne in mind that many of these cases were not secondary ulcerative intestinal tuberculosis. Those interested in the surgical side of this problem should consult Brunner's excellent monograph.

Archibald reported in 1917 the operative results in 33 cases, some of which are included in our figures. He removed the appendix in 6 cases, with great relief of all symptoms while in the hospital, though 2 died within a year from pulmonary tuberculosis. In 5 patients, he excised the bowel from the lower ileum to the transverse colon

for disease of the appendix and cecum. All had complete relief in the hospital and marked or complete relief later. Four of these died from pulmonary tuberculosis (1 had an acute obstruction relieved by operation) and 1 is alive and well. In 2 cases of resection of the cecum and colon, the relief was complete in 1, great in the other. Both died from pulmonary tuberculosis. When both small and large bowel were involved (15 cases), a palliative operation was done in 11; appendectomy was performed in 7, with partial to great relief. Five of these are dead and 2 untraced. An ileo-sigmoidostomy was performed in 4 cases with great relief at once, partial relief later. Three are dead, 1 alive. Radical operation was performed in 4, consisting of resection of the ileo-cecum in 1 and from the ileum to the transverse in 3. One died three days after operation, probably from ileus. Two had complete relief and 1, in which $6\frac{1}{2}$ feet of ileum were resected, lost all pain, but had persistent diarrhea until death. One died from ptomaine poisoning. Two cases had involvement of the colon and rectum. One had a colostomy and died after a second operation of operative shock, one year later; the other, after a resection of the cecum, and a colostomy also, had relief of symptoms but still had an artificial anus.

If we omit the 6 cases, in which only the appendix was involved, we find of the 27 remaining, 20 are dead, 5 are alive (2 are well, 1 has chronic pulmonary tuberculosis, 1 has an artificial anus) and 2 are untraced. Of those alive 3 had no pulmonary tuberculosis, 1 had a slight bilateral active pulmonary tuberculosis, and 1 incipient disease. If we exclude all those, who had no pulmonary tuberculosis, we find 18 dead and 2 alive, but 1 of them with a chronic pulmonary tuberculosis. The remaining living case was in the minimal stage. Such figures as these show how seriously handicapped is the surgeon who attempts serious operation on such patients. No doubt, in many cases the operation greatly prolonged life and gave great relief, even when cure was not effected. But the final result was that 90 per cent of the patients died from the original disease, from the complication, or, as in 1 case, from the operation.

In a later paper (1920) Archibald reported 74 cases of intestinal tuberculosis (60 of which were his own) which came to operation, with an operative mortality of 6.7 per cent and more favorable ultimate results. The 5 deaths were apparently due in 3 to a streptococcus infection, in 1 to atonic obstruction, and in 1, the only one connected with the lungs, to acute suppurative bronchitis with edema. In 10 cases the appendix alone was diseased and,

after appendectomy, all were relieved of their appendiceal symptoms. In 20 cases, the cecum and right half of the colon were involved; 7 are alive, 12 dead and 1 untraced. In 4 cases, the whole colon to the sigmoid was involved and 2 are living and 2 dead. In 32 cases, both large and small bowel were involved and of these 23 are dead, 5 living and 4 untraced. Four cases had the rectum as well as the colon diseased and 3 of these are dead. Seven cases had only the small bowel involved and 4 are living and 3 dead. The mesenteric glands were alone involved in 5 cases, of which 2 are dead.

In 31 cases resection was performed, usually most of the right colon being removed. The operative mortality was 13 per cent. Excluding 12 cases in which the operation was too recent or the whole of the disease was not removed, 19 cases are left, of which 7 are alive and 12 dead, usually from pulmonary tuberculosis. Short-circuiting was performed in 10, of which 6 are dead, 3 alive and 1 untraced.

The prognosis, Archibald believes, depends in large part upon the lungs and he divides his cases into four groups: (1) Those without any pulmonary tuberculosis discoverable by physical examination, 15 in all, of which 8 are alive, 3 dead, and 4 untraced. (2) Eight cases with early pulmonary disease, of which 5 are alive, 2 dead and 1 untraced. (3) Twenty-six cases with extensive chronic, slowly progressing pulmonary disease, of which 12 are alive and 14 dead. (4) Thirty cases with extensive and progressing disease of which 3 are alive, 24 dead and 3 untraced.

Archibald notes two chief contraindications to operation, extensive and progressive pulmonary involvement and extensive disease of the small bowel. If more than 3 feet of the small bowel above the ileo-cecal sphincter are involved, it is unwise to attempt operation. Resection should be attempted only if the lungs are in a favorable condition; otherwise a bilateral mucous fistula may be established.

Paterson has reported a series of 22 cases of intestinal tuberculosis, which were treated surgically. Of these 6 were alive and well, 4 alive and improved, 4 alive an unimproved and 8 dead. Death was hastened by operation in 3. Archibald's figures include some of these cases.

Stewart submitted 7 patients with pulmonary and intestinal tuberculosis to operation. One patient was so unfavorable that he died within a short time of the operation. Two had unilateral exclusion for extensive disease, 1 of whom recovered and 1 showed

definite improvement. Two, with extensive disease, were simply opened and closed. Another, with extensive disease, is making a good recovery after removal of a troublesome looking appendix.

TABLE LI.—RESULTS FROM OPERATION IN SECONDARY INTESTINAL TUBERCULOSIS. (Archibald's figures.)

Pulmonary tuberculosis.	Total	Living.	Dead.	Untraced.
1. None	15	8	3	4
2. Early	8	5	2	1
3. Extensive chronic slowly progressing	26	12	14	0
4. Extensive to progressive	30	3	24	3
Total	79	28	43	8
Intestinal lesion:				
Appendix	10	6	4	0
Cecum, colon and one-half rectum	20	7	12	1
Whole colon and sigmoid	4	2	2	0
Large and small bowel	32	5	23	4
Rectum and colon	4	3	1	0
Small bowel	7	3	4	0
Mesenteric glands	5	3	2	0
	82	29	48	5

In his recent work on the colon, Gant in speaking of the surgical treatment of intestinal tuberculosis mentions enterostomy, appendicostomy, cecostomy, appendicecostomy, ileo-cecostomy, colostomy, intestinal exclusion and resection. He speaks highly of appendicostomy, followed by irrigation, which he has employed in weak patients before other operative procedures. Better results, he says, can be obtained by such lavage in one month, than by many other methods in six months. It can be continued for six months.

He quotes the figures of Hartmann:

TABLE LII.

	No. of cases.	No. of deaths.	Percentage.
Partial resection of cecum	9	1	11
Resection with end-to-end anastomosis	78	19	24
Resection with side-to-side anastomosis	31	5	16
Resection at "two times"	10	3	33
Ileocolostomy	29	4	14
Unilateral exclusion	9	1	11
Bilateral exclusion	22	2	9
End-to-end anastomosis	19	3	15
Multiple operations	22	8	36
Total	229	46	20

Also those of Conrath:

TABLE LIII.

	No. of cases.	No. of deaths.	Percentage.
Resection	58	11	19
Resection intestinal wall	6		
Entero-anastomosis	10		
Enucleation	8	2	25
Exploratory laparotomy	4		
	86	13	15

His conclusions are: That the operative mortality is no higher than that for similar non-tuberculous conditions; that through-and-through colonic washings are good; that short-circuiting relieves symptoms and should be carried out, if no excision is possible; that the length of bowel removed within certain limits has no effect on the mortality; that simple laparotomy is of no help; and that general measures should never be omitted.

Hofmeister used excision in 50 cases of ileo-cecal tuberculosis and 34 healed. Kocher had 16 recoveries in 18 cases and Körte 11 operations and no deaths.

Alglave, in 1904, published tables of 50 resections of the ileo-cecum with 7 deaths (14 per cent) and of 29 short-circuiting operations with only 2 deaths (7 per cent).

Coffey, in 1921, reported a series of patients on whom he used short-circuiting for intestinal tuberculosis nine or ten years previously and who at present are in good health.

Holländer has reported a case where the patient, a diplomat, had severe intestinal disturbances and persistent vomiting, due to ileo-cecal tuberculosis. On operation, it was impossible to do an anastomosis between the ileum and the transverse colon, but on account of the greatly dilated bowel, he fixed the cecum in a normal position. The patient lost practically all of his symptoms. He thinks that the fixation had something to do with giving the cecum rest and aiding in recovery.

Lemon is of the opinion that the intestinal tuberculosis is "influenced by the state of activity and the degree of involvement of the lungs. For this reason the condition in the lung completely controls the prognosis of any surgical procedure that may be contemplated for the relief of intestinal tuberculosis."

Our results from excision and from short circuiting were not what we had hoped for. Some of the patients stood the operation badly, which is a severe one, attended often with considerable shock, and some of those submitted to short-circuiting had more pain and

diarrhea than before. The next operation used was colostomy or ileostomy, and we are convinced that it prolonged the lives of some patients. One case, a veteran of the recent war, was desperately ill with advanced pulmonary tuberculosis with cavities, and intestinal tuberculosis. A short circuit of the ileum into the pelvic colon gave him some relief. But he began again to do poorly and a colostomy was performed. The result for a time was all that could be asked. When he was again at a standstill, artificial heliotherapy wsa helpful. He became fat, full-blooded, but the colostomy opening troubled him and he wished it closed. A third operation was performed and he died in the hospital. Our results from surgical measures are as follows:

Among the cases treated by us in Saranac Lake Village 14 were subjected to operation and 10 are dead. One, however, was apparently a case of primary intestinal tuberculosis. If we except this case the mortality was 75 per cent. Of the 3 living cases, 1 had only an appendectomy, refusing further operation, and 2 were early and had complete excisions. Among those dead, 3 had excisions, 2 simple laparotomies, 2 short-circuiting, 2 appendectomies.

Of the 5 Trudeau cases undergoing operation for resection, 4 are dead and 1 alive. Combining these figures we get 14 dead (78 per cent) and 4 living.

From our experience, we are led to agree with Professor Archibald that the safest surgical measure at present is to bring each end of the affected loop or portion of the bowel, out through the abdominal wall, making in this way two mucous fistulæ, and anastomose the remaining normal loops. The intestinal contents would, by this method, pass over no diseased portion of the bowel. If the disease is very localized and the patient in good shape and can stand it, excision is by far the best method but never free from danger.

In one or two cases simple laparotomy seemed to exert some beneficial influence but in many others it apparently had no influence upon the course of the disease.

It is conceivable that under certain conditions perforation in intestinal tuberculosis might be treated successfully by surgical interference, which has been so helpful in many cases of stenosis. No cases needing surgical interference for stenosis, abscess or peritonitis has come within our notice. One patient, whose ileo-cecal region had been successfully excised four years previously and a lateral anastamosis performed, suffered a perforation at the end of the ileal "stump," followed by acute general peritonitis. An uneventful recovery followed a second operation for obstruction.

MEDICINAL TREATMENT.

The use of medicinal measures, in the treatment of the symptoms of intestinal tuberculosis, extends back to antiquity and the list of remedies, already suspiciously long, increases each year. It is possible to mention only a few, and it seems best to group them as far as possible under the symptoms for which they are used.

Diarrhea.—Diarrhea shares with pain the first place in frequency and importance among the symptoms of intestinal tuberculosis. It often first draws attention to the bowel condition and, although at times alternating with constipation, usually persists in fatal cases to the end. The mere physical exercise of using a commode or bedpan ten or more times a day emphasizes the fact that it needs to be controlled as soon as possible to preserve the strength of the patient. The diet should be carefully regulated and the fluids may have to be restricted (see p. 230). Rest in bed should be insisted upon and the abdomen kept warm.

Castor Oil.—In some cases castor oil, even when nauseating, yields excellent results in doses of $\frac{1}{2}$ to 1 ounce. Such patients experience considerable relief and increased comfort for several days, or even for weeks, following its administration. These patients should be given the oil at regular intervals but unfortunately even in such patients, it seems after a time to lose its effect.

It may be of interest to mention here a case recorded by Rogers, where a woman, single, aged twenty-eight years, who had suffered from diarrhea with bloody stools and had had a temperature of 101.3° F. from March 22 to May 9, grew steadily worse, with continuing hemorrhages and vomiting, in spite of many remedies. On May 9th, she was too weak to turn in bed and castor oil was given every hour in drachm doses. She improved almost at once and made a good recovery, in spite of a slight relapse.

Calcium.—In many cases it is wise to give calcium chloride intravenously, if the bowels, following the administration of castor-oil and restriction of diet, are not checked immediately. It is probably the best remedy we possess today. It may check the diarrhea, temporarily or even permanently, and so aid in conserving strength while other measures are applied.

The usual dose is 5 cc. of a 5 per cent solution, given once or even twice a week, for four or five or more doses, or until it ceases to work. Many use larger doses. Rist and his associates say that if the use of a 10 per cent solution does not relieve, they use 2 to 4 cc. of a 50 per cent solution. Merklen and Dubois-Roquebert have used

the same dosage with good results. The use of such large doses is not unattended with danger.

D. Mandl, who first suggested its use, thought that calcium chloride affected the vegetative nervous system and so reduced peristalsis. He quoted Soetbeer, and Krieger and Tobber, who found that the inflamed intestine secreted calcium badly. Mandl gave it by mouth, but found it worked better by enema, and best by intravenous injection, using at first a 3 per cent and, later, a 5 per cent solution. It acted quickly, often after two or three doses. Later, he gave 10 cc. and called attention to the pain, or even necrosis, that occurred when a few drops escaped into the tissues. He found it often relieved the intestinal pain as well as the diarrhea and reported the treatment of 7 cases of intestinal tuberculosis with good results.

Saxtorph used a 5 per cent solution in 6 patients, 3 of whom were freed from all intestinal symptoms for a long time, but the remaining 3, all with far advanced pulmonary tuberculosis, showed no response.

Fishberg found 5 cc. of a 5 per cent solution effective only in early cases of intestinal tuberculosis, where the pain also may be abolished. In the later stages it may restrict the number of stools. He suggests that it acts upon the nervous system and decreases peristalsis. He reports 7 cases with success in 5.

Ringer and Minor treated 30 cases of tuberculous diarrhea with 5 to 10 cc. of a 5 per cent solution of calcium chloride. Eight did not have extensive pulmonary tuberculosis, in 2 the diagnosis of "tuberculous diarrhea" was doubtful, and, in 16, the pulmonary tuberculosis was advanced and hopeless and the treatment only palliative. Their results were as follows: Better 9, worse 1, dying 1, dead 16, untraced 3. They spaced the intervals and numbers of doses according to the symptoms. One patient had fifty-two doses, 1 fifteen, 1 eleven, 1 seven, 1 six, 2 five, 2 four, 3 three, 16 two and 3 one. Of the 16 cases with two doses, 13 had no benefit and the treatment was stopped. In 3, the symptoms ceased and no more doses were given. One case was treated for twenty months (fifty-two doses) and the dose raised to 9 cc., to which he reacted. Some of the patients had a strange feeling of heat over the body which began in the throat. Two or three felt faint, but were nervous patients. Rist noted temporarily in some patients an intense pain in the shoulder, a metallic taste, a red face and a feeling of heat in the head.

In a study of 70 cases of intestinal tuberculosis treated with intravenous injections of calcium chloride (5 cc. of a 5 per cent

solution, twice a week for one year, if necessary) at the Fitzsimons General Hospital in Denver, E. H. Roberts saw relief or improvement of their gastro-intestinal symptoms in 80 per cent (20 per cent with apparently complete relief); negative results in 14 per cent; and in 3 per cent, 2 cases, healing in 1, in spite of death from pulmonary tuberculosis, marked improvement in another. The results from heliotherapy, which apparently he limits to the abdomen, were not so good and the use of calcium chloride has been adopted in the hospital as the standard treatment, but dietetic and heliotherapeutic measures are used as well.

The explanation of the action of calcium is said by Pellé to be due to hemoclastic shock. He found that the leukocytes decrease 50 per cent, the blood-pressure falls and the clotting time decreases. Starkenstein says calcium influences the functions of the whole organism in its capacity for reaction, and limits inflammation. Mandl, as noted previously, thinks it acts through the nervous system. Some hold calcium forms an insoluble compound with the surface proteins while it is being excreted through the intestinal mucosa.

In certain cases, calcium salts administered by mouth are unquestionably of value and may be given as lime water in milk, aromatic chalk powder (10 to 60 gr.), calcium lactate (5 to 10 gr. every four hours), or in the following combination, which has proved very helpful: calcium phosphate and calcium carbonate, of each 1 ounce, with or without bismuth subnitrate, $\frac{1}{2}$ ounce: *Dose:* A teaspoonful, after meals. Calcium carbonate alone may be used for hyperacidity, if it is suspected that sodium bicarbonate increases the diarrhea.

It is still an open question whether calcium salts, given by mouth, are of any avail, when they fail by intravenous injection.

Early in the disease, the diarrhea will often yield to simple remedies, such as are usually employed for slight attacks of acute diarrhea. Agar-agar may render the stools less watery. When the patient is subjected to heliotherapy, often no medicinal treatment is required.

Many patients, however, in whom extensive ulcerative changes have occurred in both lungs and intestines, seek treatment, where heliotherapy affords only temporary, palliative relief, if, indeed, any. For these patients many remedies must be tried one after another until one is found that gives some relief.

Bismuth.—The bismuth salts have been widely and often successfully employed. The benzoate, hydrate, subnitrate (15 gr. t. i. d.); subgallate (dermatol), citrate, betanaphtholate, salicylate,

subsalicylate, gallate, tannate, sulphocarbolate, etc., have been used. Other preparations include airal (bismuth oxyiodide), thioform (bismuth dithiosalicylate), orphal (B-naphtholate), xeroform (tribromphenolate).

These salts are often combined with various astringents.

Astringents.—The astringents are often helpful, and many depend for their action upon their tannic acid contents. Substances containing tannic acid usually affect the stomach slightly but with the alkaline reaction of the intestine, they precipitate any protein with which they come in contact, and so protect the mucous membrane, which is less stimulated; peristalsis is thus lessened, and absorption aided by longer contact but slowed for the reason given above. The mucus may be lessened which also helps to retard the passage of the contents. The various microörganisms may be somewhat affected and fermentation hindered.

These substances may be given in the form of a drink, such as acorn cocoa, acorn coffee, red wine, which are pleasant, mild and not unpalatable. Catechu, gambir, krameria, radix, and kino, as simple or compound tinctures, 15 to 30 drops, every four hours, may relieve in different cases. Kino as a powder (10 gr. with bismuth) or in a solution (fluid extract coto., tr. cardamom co., of each 1 drachm, mix and triturate slowly with mucilage of acacia, 3 drachms, simple syrup, 2 drachms; and finally water to 6 ounces: *Dose:* Tablespoonful, three or four times a day). Cotoin, in doses of $\frac{3}{4}$ gr. in sugar of milk, every three hours, has been used successfully. Though cotoin is derived from kino, it is said not to contain tannic acid but to act, like adrenalin, through the sympathetic nervous system, to lessen the pendulum movements of the intestinal loops. Eldoform (tannin combined with yeast) may aid.

While witchhazel and hematoxylin have been employed, more recently several synthetic compounds have come largely into use, which are said to be inert until acted upon by the pancreatic juice. These include tannigen (5 to 10 gr., three or four times a day), tannalbin (5 to 8 gr.), tannoform (5 to 10 gr.), tannocol (5 to 10 gr. frequently), tannopin (5 to 10 gr.) tannismuth (5 to 10 gr.).

Other forms of astringents, more often employed formerly than now, include zinc oxide (1 to 3 gr.), copper sulphate ($\frac{1}{4}$ to $\frac{1}{2}$ gr.), lead acetate (1 gr., as a pill), purple willow (℥j, 4 or 5 i. d., in syrup).

Inert Powders.—Certain inert powders seem in some cases to influence favorably the diarrhea, either by coating and protecting the mucous membrane, or by absorption or adsorption of gas and bacteria. Among them may be mentioned animal charcoal in

large doses (½ ounce in milk or sweetened arrowroot, three times a day), kaolin (a drachm every three or four hours), and talcum purificatum.

Iodoform (¼ gr.) and beechwood creosote (♏j) in a capsule, after meals, as recommended by Yeo, are helpful in some cases. In cases with distention, 1 drop of creosote in a tablespoonful of lime water, just before meals, may aid. Methylene blue (2 gr.) with sugar, three or four times a day, may aid. In some instances the following may help when given before meals as a powder: Bismuth subgallate 5 gr., betanaphthol benzoate ⅕ gr., resorcin 7½ gr. Ipecacuanha (½ gr.) may be combined with bismuth and sodium sulphocarbolate (10 gr.) in chloroform water with mucilage of acacia. Guaiacol carbonate may be of aid. Acriflavin may be tried, giving a tablet (½ gr.), two or three times a day.

Enemata.—Enemata of warm physiological salt solution, with or without 10 to 20 drops of laudanum (tincture of opium), when given slowly, and under little pressure, are grateful to some, very exhausting to other patients. The fluid should be withdrawn in large part through a colonic tube, which saves greatly the strength of the patients.

Hurst recommends albargin (silver nucleate) in enemata, as very effective in ulcerative colitis. Gant, who recommends colonic washing through an appendicostomy opening, suggests as irrigations: hot water (100° to 115° F. very soothing), and silver nitrate, (30 gr. to a pint of water), three times a week, to be followed by normal salt solution. As the patient improves, he substitutes the following: Boracic acid, 3 per cent; ichthyol, balsam of peru, 2 per cent; potassium permanganate, 1 per cent; argyrol 5 per cent. These strengths should be increased or decreased, according to the symptoms. One can use warm olive oil, or other forms of oil, alone, or with bismuth, aristol or salol. Others have suggested salicylic acid 1 to 1000; tannin, 5 to 1000; silver nitrate 1 to 2000. Oil in amounts of 4 to 8 ounces, with or without 1 per cent creosote (Fleiner) have also been employed.

Tannigen (5 per cent), alum (1 per cent), argentum colloidale (0.5 to 5.0 to 1000) have been tried. One to 2 drachms of bismuth subnitrate in 1 to 2 pints of warm water have given beneficial results. The water should be withdrawn or passed in one-quarter of an hour. In using any of these enemata, a cleansing enema may be used first, unless the diarrhea has been marked.

Opiates.—In many patients with advanced lesions in both lungs and intestines, all the remedies mentioned may fail and resort must

then be had to opiates. A combination of Tully powder (pulvis morphinæ co.) and salol, 2½ gr. each, given after each meal, was a favorite remedy of Dr. Trudeau's and will be found very satisfactory in many cases. The dose may have to be increased in a few cases. The starch and opium enema (thin starch, ℥ij and tincture of opium ℥ss) may also be tried, when other measures have failed, and it often affords great comfort. Lead and opium pills [acetate of lead and powdered opium 1 gr. each, with or without extract of henbane (2 gr.), one, two or three times a day] may be tried. Sulphate of copper (¼ gr.) may be combined with opium. Opium suppositories are useful at times (dose, ¼ gr. extract of opium). The deodorized tincture of opium is usually well borne. Dover's powder (pulvis ipecacuanha co., 5 to 10 gr., every four hours) is often valuable.

Of all the opiates, none is safer to put into the hands of the nurse or family than paregoric (camphorated tincture of opium), which can be given in ½ or 1-drachm doses, three or four times a day—a dose not usually required. It must not be forgotten that many patients feel better when the bowels are loose.

Constipation.—The constipation which alternates with diarrhea should be treated with mild laxatives or mineral oil (which can be continuously administered). Calomel should be avoided. Castor oil can be safely used.

Pain.—Pain is usually quickly relieved by heliotherapy or calcium salts. If it persists, local applications should be first tried. The peristaltic unrest, described often by patients as grumbling in the abdomen, is at times relieved by a hot wet compress after meals. For the more severe pains, hot fomentations, linseed poultice, antiphlogistine, turpentine stupes, hot water bottles or electric pads may give relief. The application of Baumé analgesic (Bengué) to the abdomen, which should be immediately covered, is often grateful. An ointment, somewhat similar, of equal parts of methylsalicylate and menthol, up to 10 parts, lanolin 12 parts, may be rubbed in (use a piece the size of a hazel nut) and covered with flannel. It is most important in these cases to avoid all exposure of the abdomen to cool air. Chilling of the feet may initiate an attack of pain. Schmidt thinks cold may relieve while heat may increase pain.

Pottenger thinks atropin is important in relaxing spasm and relieving colicky pain. Acetyl-salicylic acid, hyoscyamus, or Hoffman's anodyne may be tried. In some instances resort to opiates must be had and injections of pantopon or halogen may be tried first.

Hemorrhage.—The treatment of intestinal hemorrhages varies in no way from treatment of the same condition in other diseases. Calcium chloride should be injected at once. The use of castor oil has been mentioned (see p. 220). Fibrogen (Mills) may aid.

In a case of hemorrhagic colitis, Hayward used sour milk prepared with bacillus bulgaricus and incubated for forty-eight or more hours, for irrigation of the bowel through an appendicostomy opening, two or three times daily. He used 1 quart and kept it up a month. Marked improvement resulted.

Bacterial Treatment.—The bacillus bulgaricus in milk has proved of benefit in relieving symptoms in some patients and, more recently, Morriss has prescribed bacillus acidophilous milk with interesting and helpful results. He gave 1 quart of milk daily, together with 50 to 100 grams of lactose, which he had to reduce at times on account of cramping pain. Apparently only a few of his cases had tuberculous colitis but in 6, who suffered from diarrhea, this symptom was relieved.

Brachat recommends the administration of a bolus, containing $\frac{3}{4}$ gr. of chloramine-T in $37\frac{1}{2}$ gr. of lanoline, two before each meal.

Nausea and Vomiting.—Nausea and vomiting usually quickly subside under heliotherapy. Here again calcium chloride is often of considerable benefit.

General Digestive Disturbances.—The treatment of gastric disorders in pulmonary tuberculosis has long been unsatisfactory as, in large part, they are reflex conditions from the underlying intestinal tuberculosis. As long as the intestinal condition is unchanged the symptoms persist. As the intestinal tuberculosis improves the gastric symptoms abate. The usual remedies should be given a trial.

We would like to state in conclusion that, we feel some patients with early intestinal tuberculosis will no doubt recover under any and all forms of treatment, but just as it is impossible to say which patients with early pulmonary tuberculosis will recover without ceasing work, so it is impossible to say which of the patients with this complication will recover without any local treatment. It seems needless to add that patients who have lost all powers of response, who are in terminal stages, can only rarely be snatched from the gaping jaws of death.

CHAPTER XXVIII.

CONCLUSIONS AND SUMMARY.

A.—GENERAL.

1. The most frequent complication of pulmonary tuberculosis is intestinal tuberculosis. It is found in from 50 per cent to 80 per cent or more of all autopsies done on patients dead of pulmonary tuberculosis.

2. Twenty of 361 consecutive routine cases at the Trudeau Sanatorium had definite intestinal tuberculosis.

3. In 1923, 90,000 persons died from pulmonary tuberculosis, in the registration area of the United States. Of these, 40,000 to 60,000 had also intestinal tuberculosis.

B.—DIAGNOSIS.

1. The present status of the diagnosis of intestinal tuberculosis is comparable to that of pulmonary tuberculosis twenty-five years ago, when an early diagnosis aroused comment.

2. The heretofore "usual" symptoms of intestinal tuberculosis are those that occur in the more advanced stages of the disease, namely, persistent diarrhea, abdominal pain, tender points in the abdomen, with or without rigidity, in the absence of an acute abdominal condition.

3. Tubercle bacilli in the stools are of little diagnostic aid, as they occur in 85 per cent to 95 per cent of all patients with tubercle bacilli in the sputum.

4. Suggestive symptoms of beginning intestinal tuberculosis include, among others, any digestive disturbances, marked constipation, failure of the pulmonary condition to improve, an irregular temperature with subnormal fluctuations, and possibly decrease of pulmonary symptoms when the patient is not doing well, alternating constipation and diarrhea, and marked nervousness.

5. As the symptoms and abdominal examination are so often negative and clinical diagnosis so uncertain, we must turn to the

study of the barium meal by roentgen-rays to exclude intestinal tuberculosis.

6. General hypermotility, with complete or nearly complete emptying of the colon in twenty-four hours; failure of the cecum or of the ascending colon and hepatic flexure to retain the barium; or the presence of spasm or filling-defects (irregular contour, lack of haustrations); or of confirmed segmentation, with or without dilatation of some coils of the small bowel; ileal stasis; and gastric retention are the essential points when the intestine is studied at the third to the tenth hours, and again at the twenty-fourth hour. These findings indicate ulceration only, but when pulmonary tuberculosis is present, tuberculous ulceration can be safely diagnosed.

7. The barium enema usually confirms the fact that the cecum or other portions of the colon may fail to receive or to retain the barium.

8. No moderately or far-advanced case of pulmonary tuberculosis can be assumed to have been thoroughly examined unless a roentgen-ray study of the bowel has been made.

9. This is the only method which will diagnose intestinal tuberculosis in its incipiency or exclude it at any stage.

C.—TREATMENT.

1. **Medicinal Treatment.**—Medicinal treatment, when diarrhea is absent, is of little avail. Calcium chloride intravenously may aid markedly the diarrhea, and even bring about its permanent cessation. Drop doses of creosote in a capsule, with $\frac{1}{4}$ gr. of iodoform, may be tried after meals. Salol and pulvis morphinæ compound, $2\frac{1}{2}$ gr. of each, every four hours, may have to be resorted to in terminal cases.

2. **Surgical Interference.**—The roentgen-ray technic may not reveal the whole extent of the involvement. Patients with advanced pulmonary tuberculosis do not stand operations well and should not be operated upon, nor should those with advanced intestinal lesions, except to relieve symptoms. In early, localized cases, if surgery be resorted to, excision is the operation of choice, but it may be necessary to short-circuit, which in advanced cases may make the condition and symptoms worse. Exclusion of the affected portion of the intestine from the intestinal tract, bringing the two ends of the affected portion into the abdominal wall, thereby forming two mucous fistulæ, is probably the operation of choice in most cases.

3. The ultraviolet-rays and natural heliotherapy frequently relieve the symptoms and bring about recovery. Inasmuch as a few cases recover without any treatment, judgment of their true value is difficult. They should be used in all cases whether or not treated surgically.

4. When the desired results are not obtained by heliotherapy, roentgen-ray treatment of the intestinal tract should be carefully given or other measures employed.

5. Many cases make a good recovery both clinically and radiographically.

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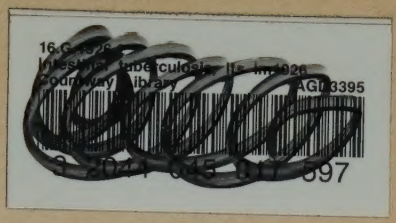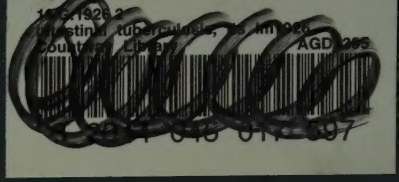
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